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Research Article

Effect of Gabapentin on Pressor Response to Laryngoscopy and Tracheal Intubation: A Double Blind Randomized Placebo Controlled Study

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(Received July, 2012) (Accepted October, 2012)

Abstract:

The effects of gabapentin on arterial pressure and heart rate were compared at induction of anaesthesia and at tracheal intubation in a prospective randomized double blind study. Fourty patients of American society of Anaesthesiologists (ASA) physical status I undergoing elective surgery were divided in two groups of twenty each. Twenty patients received oral placebo (Group P), and 20 patients received 900 mg of gabapentin (Group G), 2 hours prior to induction of anaesthesia. Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Heart rate (HR) were recorded one minute before and after induction of anaesthesia, immediately after intubation and 1,3,5 and 10minutes after intubation. Changes in SBP were statistically insignificant in both the groups. In the gabapentin group, at 0 and 5 minutes, the DBP was significantly less than the placebo group ($p<0.05$). There was a significant decrease in heart rate in Group G as compared to Group P ($p<0.05$).Pre medication with 900 mg gabapentin, 2 hours before induction of anaesthesia attenuates the tachycardia associated with laryngoscopy and intubation but not the pressor response completely.

Key Words: Gabapentin, Laryngoscopy, Intubation, Haemodynamic responses

Introduction:

Tracheal intubation provokes a marked sympathetic response manifested as tachycardia and hypertension, which is potentially deleterious in some patients. Apart from hypertension and tachycardia, dysrhythmias and myocardial ischemia can occur (Brandt, 1996). The mechanism for these reflex cardiovascular changes is unknown, but the drop may be a result of reflex sympathetic action perhaps involving the baroreceptor system provoked by stimulation of the epipharynx and laryngopharynx.

Several techniques have been proposed to prevent or attenuate the haemodynamic responses following laryngoscopy and intubation, such as deepening of anaesthesia, omitting cholinergic premedication, pre-treatment with vasodilators such as nitroglycerine (Mikawa et al, 1992), beta blockers (Coleman et al, 1980), calcium channel blockers (Puri & Batra, 1988; Nishikawa & Namiki, 1989) and opioids (Maguire et al, 2001; Dahlgren & Messeter, 1981).

Recently gabapentin has been used in randomised controlled trials to treat acute postoperative pain and to reduce postoperative opioid requirements (Fassoulaki et al, 2006 ; Thomson et al, 1989). In these studies with gabapentin, it was noticed that patients remained haemodynamically stable. Thus, we found it worthy of evaluation as to whether gabapentin has an effect on changes in blood pressure and heart rate during laryngoscopy and tracheal intubation or not.

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Material and Methods:

This prospective, randomized, double blind and placebo controlled study was performed on forty patients undergoing elective surgery under general anaesthesia. Patients with ASA grade I in the age group of sixteen to sixty years were included in the study. Patients were divided into groups of 20 each. Group P received oral placebo in the form of 3 sugar filled in emptied gapapentien capsule two hours before the surgery and Group G received three gabapentin capsules (300 mg each) were 2 hours before Surgery. Patients were familiarized with verbal rating scale (VRS) for sedation. Ranitidine 150mg was given the night before surgery at 10 pm to all patients.

Exclusion criteria were: anticipated difficult intubation, ASA physical status II or above, history suggestive of hiatus hernia and gastroesophageal reflux, patients with body weight >20% of ideal body weight, consumption of anti hypertensive, sedatives, hypnotics and antidepressant drugs; patients with nervous system disorders and history of drug hypersensitivity.

Informed consent was taken from all patients after approval from ethical committee.

Anaesthetic Technique:

In the operation theatre, intravenous ringer lactate solution was started. All patients received intravenous ondansetron 0.1mg/kg and tramadol 2.0 mg/kg 10 minutes before induction of anaesthesia. Standard monitors were attached like heart rate, blood pressure, ECG and pulse oximeter.

After tracheal intubation, end tidal carbon dioxide monitoring was initiated, paracetamol infusion was given over 15 minutes with 15 mg/kg body weight and injection diclofenac sodium aquous was given intravenously with a dose of 1 mg/kg body weight. All patients were preoxygenated for 3minutes. Anaesthesia was induced with oxygen, nitrous oxide, and propofol 2mg/kg of body weight. Vecuronium 0.1mg/kg was used to facilitate tracheal intubation. All intubations were performed by experienced anaesthesiologist. The duration of laryangoscopy and intubation was limited to the minimum possible time of 30 seconds and was done in a single attempt. Anaesthesia was maintained with nitrous oxide 66%, oxygen 33%, halothane 0.5% and vecuronium.

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were recorded one minute before and after administration of the intravenous anaesthetic agent. The next reading was taken immediately after intubation and cuff inflation and was labelled as the 0 reading. Subsequent readings were taken at 1,3,5 and 10 minutes. Continuous intra operative monitoring of heart rate and blood pressure was done at every 5 minutes. Neuromuscular block was reversed with neostigmine 0.05mg/kg and atropine 0.02mg/kg intravenously. After tracheal extubation and on awakening from anaesthesia, patients were assessed for postoperative sedation using an 11 point VRS (with 0= no sleepiness to 10= almost asleep) at 0, 1, 6, 11 and 24 hours.

Statistical Analysis:

Data was analyzed on computer software. Characteristics of the patients of the two groups and changes in SBP, DBP and HR between the two groups were compared with independent *t*- tests. The sedation score was compared using the mann witney (non parametric) test. Somnolence and dizziness amongst the patients in the two groups were analyzed using the *chi-square* test.

Results:

It was found that the two study groups were comparable with respect to age, sex, height, weight and duration of surgery (Table I). The mean age was 42.55 yrs in the gabapentin group (group G) while it was 42.7 yrs in the placebo group (group P). Similarly, the mean height was 149cms in the group G and 150.2cms in group P. The mean weight also did not differ much; 54.75kg v/s 52.35kg. The duration of anaesthesia was 123.5mins in group G and 128 mins in Group P.

The baseline values of SBP were comparable (127.70mmHgvs 126.80mmHg) in both the groups (Table II). After adiministering propofol, there was a fall in SBP followed by a rise after laryngoscopy and intubation in both the groups . The trend of fall towards the base line values was similar in both the groups at 0,1,3,5 and 10 mins after intubation.

The sedation scores in the present study were significantly higher in the group G at 0 and 1 hour in the postoperative period (Table V).

Table I: Patient characteristics in each group.

	N	Group	Mean± S.D.	*p*-value
Age (yrs)	20	G	42.55±7.38	0.993
		P	42.7±8.48	
Height (cms)	20	G	149.0±3.35	0.35
		P	150.2±4.36	
Weight (kg)	20	G	54.75±8.94	0.37
		p	52.35±7.74	
Duration (mins)	20	G	123.5±16.14	0.32
		P	128±10.43	

Discussion:

Tracheal intubation is a noxious stimulus, tending to provoke a marked sympathetic response manifested as tachycardia and hypertension which is potentially deleterious in some patients. Various agents effectively attenuate this response, including anaesthetics, analgesics, adrenergic blocking agents and vasodilators. This has been a fertile area for clinical investigation, spawning numerous studies of the various techniques which might be expected to modify the haemodynamic response to intubation. Some patients unquestionably require careful haemodynamic control during induction of anaesthesia and intubation. Even a transient hyperdynamic response may cause serious complication in patients with symptomatic aortic aneurysm, recent myocardial infarction, cerebral

Table II: SBP before and after intravenus (i.v.) induction agent and at 0,1,3,5, and 10 mins after tracheal intubation and Cuff inflation in the gabapentin and placebo group.

Group		Before i.v.	After i.v.	0 min	1 min	3 min	5 min	10 min
G	Mean	127.70	117.8	128.7	119.2	112.7	107.6	104.0
n=20	± SD	10.67	10.86	11.116	10.462	11.526	10.515	11.079
P	Mean	126.80	114.65	138.65	124.25	114.85	109.25	105.90
n=20	± SD	11.423	11.735	20.630	18.157	12.704	11.026	10.422
t-value		0.25	0.88	1.89	1.065	0.548	0.471	0.561
p-value		0.798	0.384	0.67	0.293	0.578	0.641	0.580

Table III: DBP before and after intravenus (i.v.) induction agent and at 0 , 1, 3, 5 and 10 min after tracheal intubation.

Group		Before i.v.	After i.v.	0 min	1 min	3 min	5 min	10 min
G	Mean	81.00	74.90	85.45	78.32	73.05	68.21	67.21
n=20	±SD	7.248	7.174	11.190	11.480	9.554	8.237	7.729
P	Mean	85.25	77.70	94.50	83.75	77.40	75.05	72.90
n=20	±SD	7.853	8.355	14.562	13.490	12.927	11.954	10.427
t-value		1.791	1.146	2.216	1.376	1.228	2.104	1.97
p-value		0.083	0.263	0.034*	0.185	0.242	0.045*	0.062

Table IV: HR before and after intravenus (i.v.) induction agent and at 0,1,3,5,10 mins after tracheal intubation and Cuff inflation in the gabapentin and the placebo group.

Group		Before i.v.	After i.v.	0 min	1 min	3 min	5 min	10 min
G	Mean	89.70	83.00	97.05	92.50	87.90	82.35	77.50
n=20	±SD	13.800	12.465	17.191	14.490	13.676	12.067	8.382
P	Mean	94.55	89.40	110.85	104.90	99.10	92.85	84.45
n=20	±SD	16.491	12.882	14.317	13.058	12.649	13.136	11.732
t-value		1.012	1.606	2.608	2.858	2.702	2.64	2.151
p-value		0.32	0.119	0.009*	0.007*	0.011*	0.012*	0.038*

Table V: Sedation scores in the post operative period at 0,1,6,11,24 hours after surgery.

Group		0 Hr	1 Hrs	6 Hrs	11 Hrs	24 Hrs
G	Mean	3.20	2.80	1.40	1.00	1.00
n=20	± SD	1.005	1.005	.821	0.000	.000
P	Mean	1.85	1.35	1.05	1.05	1.00
n=20	± SD	.336	.489	.224	.224	.000
t-value		5.99	6.22	2.169		
p-value		<0.0001**	<0.0001**	0.074	0.317	1.000

*Significant (p<0.05), ** Highly Significant (p<0.0001)

Table VI: Occurrence of dizziness at 0,1,6,11 and 24 hours after surgery.

Group		0 Hrs	1 Hrs	6 Hrs	11 Hrs	24 Hrs
G	Present	19	19	0	0	0
n = 20	Absent	1	1	20	20	20
P	Present	9	5	0	0	0
n = 20	Absent	11	15	20	20	20
χ^2-value		11.90	20.417			
p-value		0.001*	<0.0001**	----	----	----

*Significant ($p<0.05$), ** Highly Significant ($p<0.0001$)

aneurysm, or intracranial hypertension.

Known or suspected ischaemic heart disease is by far the most common indication for modifying the haemodynamic response to intubation. The value of haemodynamic control in these patients is somewhat controversial. Myocardial ischaemia is a frequent consequence of induction and intubation especially if tachycardia occurs.

Perioperative myocardial ischaemia has been associated with postoperative myocardial infarction, and a causal relationship has been postulated (Mikawa et al, 1992; Fassoulaki & Kaniaris, 1983). Therefore, elimination of ischaemia at the time of intubation might prevent infarction. Modification of the haemodynamic response to intubaton is a laudable objective and is clearly indicated in a small subgroup of patients in whom a single hyperdyanamic episode may cause a catastrophe.

A variety of drugs have been used to control this haemodynamic response (Memis et al, 2006). Recently, gabapentin was found to be effective in attenuating the pressor response to tracheal intubation in various studies. Memis et al (2006), noticed that 800 mg of gabapentin, administered orally 1 hour before the surgery, was found to be effective in reducing the noxious stimuli to larangoscopy and intubation, thereby attenuating the hemodynamic response. Misra et al (2011), demonstrated that 900 mg of gabapentin administered orally 2 hours before induction of anaesthesia, abolished the hemodynamic response after skull pin insertion. With this background, in this study, 900 mg of gabapentin was given two hours before intubation and results were analyzed.

In the study canducted by Memis et al (2006), patient receiving 400 mg of gabapentine 1 hour prior to surgery in the operation theatre showed significant increase in blood pressure associated with tracheal intubation compared to baseline level. In comparison, there was a significant decrease in heart rate and arterial pressure after intubation at 1,3,5 and 10 minutes in patients who received 800 mg gabapentin oral and 8mg dexamethasone intravenous one hour before surgery, than in patients who received 800 mg gabapentin alone preoperatively (Kovac, 1996). In a similar study by Koc et al (2007) it was seen that haemodynamics at 1,3,5 and 10min after tracheal intubation were found to be significantly lower in patient receiving a combination of 800mg gabapentin oral and 8mg dexamethasone intravenus one hour before surgery, than in patient who received either gabapentin 800 mg or dexamethasone 8mg alone.

In the present study, the comparison of DBP readings in both the groups revealed similar finding (Table III). The baseline values were similar in both the groups i.e 81mmHg in group G and 85.25mmHg in group P. There was a precipitous fall after administration of IV induction agent. Laryngoscopy led to an increase in DBP followed by a consistent decrease towards the baseline values over the next 10 mins. Inter group comparison was significant only at 0 and 5 mins. However, in the study of Fassoulaki et al (2006), patients in the group G showed lower DBP values at 0,1,3, 5 and 10 minutes.

The baseline heart rate was again comparable in both the groups in the present study (Table IV). The same was true for the readings after administration of intravenous induction agent. There was an apparent increase in heart rate in both the groups after laryngoscopy and intubation but the increase was significantly less in group G at all times. These findings are markedly different to those of Fassoulaki et al (2006), where there was an increase in heart rate in both the groups after laryngoscopy and intubation.

Memis et al (2006), observed a significant decrease in heart rate in patients who received 800 mg of gabapentin one hour preoperatively. Patients receiving placebo and 400 mg gabapentin showed a significant increase in heart rate associated with tracheal intubation compared to baseline levels than the patients who received 800 mg gabapentin. They concluded that gabapentin 800 mg given one hour before the operation blunted the arterial pressure and heart rate increase in first 10 minutes due to endotracheal intubation. Koc et al (2007)observed that the heart rate at 1, 3, 5, and 10 minutes after tracheal intubation was significantly lower in patients receiving a combination of gabapentin 800 mg orally and dexamethasone 8 mg intravenous, given one hour before the surgery than in patients who received either drug alone.

Dizziness was experienced by a large number of patients in group G but these effects were seen only till 2 hours after surgery as later the affect of gabapentin wore off. Gabapentin has been shown to be well tolerated and effective in the management of the pain associated with post herpetic neuralgia (Parsons et al, 2004). It is assumed that adverse events occurring with gabapentin are dose related. Parsons et al (2004) observed that the three most common adverse events were dizziness, somnolence and peripheral edema. In the present study peripheral edema was not observed in any patient.

Overall, it appears that pre-operative gabapentin blunts the heamodynamic response to intubation. Single and multiple doses have comparable haemodynamic effects. Arterial pressure and heart rate responses have been shown to be greater when the duration of laryngoscopy exceeds 30 second. The mechanism of gabapentin in controlling this haemodynamic response remains unknown. Since gabapentin inhibits membrane VGCCs (Voltage gated calcium channels) it is possible that it may have a similar action to calcium channel blockers (Sarantopoulos et al, 2002). Oral administration of gabapentin 900mg, two hours prior to induction of anaesthesia, is a simple and practical method for attenuating pressure response to laryngoscopy and tracheal intubation after standard elective induction.

Conclusion:

Pre-treatment with gabapentin 900 mg two hours before the induction of anaesthesia effectively attenuates the tachycardia associated with laryngoscopy and intubation but not the pressor response completely.

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Research Article

Supernumerary Bones in the Walls of the Bony Orbit

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(Received July , 2012) (Accepted January, 2013)

Abstract:

Occurrence of supernumerary bones in the walls of the orbit especially in the medial wall and the roof has been described in the literature. Studies of the prevalence of supernumerary bones in the bony wall of the orbit are scarce in the literature. Present study was undertaken to find the prevalence of supernumerary bones in the walls of the orbit in a collection of adult Indian skulls.

In the present study three hundred and twenty six orbital walls from one hundred and sixty three skulls were examined for the presence of the sutural bones. Their location with reference to the sutures in the walls of the orbit and their size was noted. The supernumerary bones were found in 25 skulls (15.34 %) mainly in the lateral wall (11.04 %) and the roof of the orbit (4.29 %). Prevalence of such supernumerary bones in the walls of the bony orbit is of anthropological interest. Many of the bony ossicles were of sufficiently large size, enough to be visualized on lateral skull X-ray and could easily be mistaken for fracture of the bony wall of the orbit.

Key Words: Bony ossicles; Cranium; Orbit; Supernumerary bones; Wormian bones.

Introduction:

Small islands of bones called the sutural bones are known to occur along the cranial sutures or at the junction of cranial sutures on the cranial vault (Inkster, 1951; Black, 2008). The supernumerary bones on the lateral wall of the orbit don't find any mention in the standard text books of anatomy. A number of studies on the prevalence of the sutural bones of the skull among the different races of the world are found in the literature (Carolineberry & Berry, 1967; Pal et al, 1986; Gopinathan et al, 1998).

Specific studies aimed at the prevalence and other morphological features of such sutural bones are scarce in the recent literature. However, Malhotra et al (1980) have reported a prevalence of os orbitale in the roof of the orbit in a collection of Indian skulls.

Hence, an attempt has been made in the present study to ascertain the frequency and morphological features of these sutural bones occurring in the walls of the bony orbit.

Material and Methods:

This study consisted of one hundred and sixty three macerated adult skulls of unknown sex of south Indian origin which were examined from a collection of adult Indian skulls available in the Department of Anatomy, St John's Medical College, Bangalore.

In each skull, the inner surface of the walls of the bony orbit were inspected carefully for the presence of the supernumerary bones. Wherever the supernumerary ossicles were encountered, their exact location with reference to the sutures on the wall of the orbit was noted and their maximum diameters perpendicular to each other were measured to the nearest of mm with a digital caliper. The mean and standard deviations of the above values were calculated.

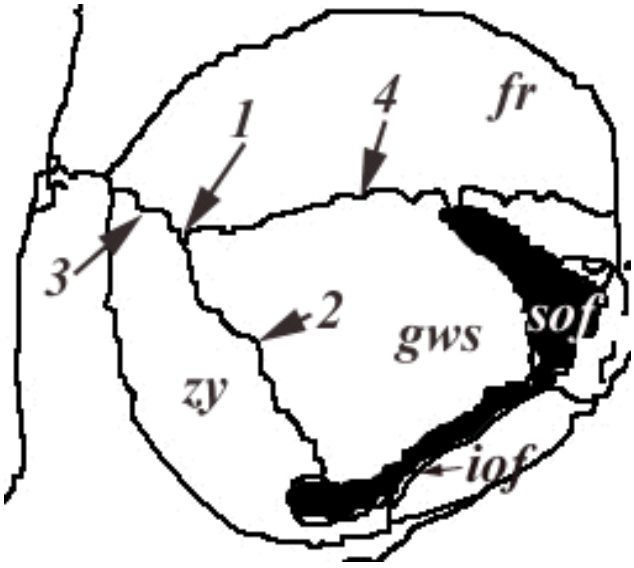

Fig. I: Diagram of the orbit showing locations where supernumerary bones were found on the lateral wall of the skull.1- junction of fronto – spheno – zygomatic sutures; 2- spheno-zygomatic suture; 3- fronto-zygomatic suture; 4- spheno frontal suture.

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Observations:

Supernumerary ossicles were found mainly in the lateral wall and the roof of the orbit. They were of variable size and shape and their occurrence in different locations is shown in the Table-I.

Ossicles in the lateral wall of the orbit:

Supernumerary ossicles were found in 18 skulls (11.04%). Only in two cases it was observed bilaterally and in all other cases the ossicles were observed unilaterally of which thirteen were of right side and three were of left side.

The ossicles were found on the lateral wall in four sites (Fig. I) viz: (1) junction of the suture between frontal, sphenoid and zygomatic bones; (2) along the spheno-zygomatic suture; (3) along the fronto-zygomatic suture and (4) along the spheno-frontal suture.

Site 1: In seven skulls a supernumerary bone was found wedged at the junction of the frontal sphenoid and zygomatic bones (Fig. II-A, C, D, E). Of these in one skull it was bilateral and in other six skull it was unilateral (5 right side and 1 left side). In one skull on the right side the ossicle occupied the full thickness of lateral wall and was visible on the floor of the temporal fossa (Fig. II-B). In another skull it was associated with a supernumerary ossicle along with the spheno-zygomatic suture.

Site-2: In seven skulls, the supernumerary ossicles were found unilaterally along the spheno-zygomatic suture (5 on right side and two on left side; Fig. II-F,G). In one skull on the right side, two ossicles were found one below the other (Fig. II-I,H) and in another skull four ossicles were found successively along the spheno-zygomatic suture extending upto the upper border of the inferior orbital fissure (Fig. III-J). In another skull a number of ossicles were found clustered within an area along the spheno-zygomatic suture (Fig. III-I).

Site-3: In only two skulls a supernumerary ossicle was found on right side only in fronto-zygomatic suture (Fig.II-F; Fig. III-M). In one of these skulls an ossicle was observed in the sites 2 and 3).

Site-4: In only one skull an ossicle was found along the spheno-frontal suture bilaterally (Fig. III-K, L).

In only one of the skulls supernumerary ossicles were found on the external surface of the lateral wall of the right orbit (temporal fossa) along the fronto-zygomatic suture (Fig. III-N).

Ossicles in the roof of the orbit:

Ossicles of varying shape and size were found in the roof of the orbit of seven skulls (4.29%). In one skull tiny ossicles of roughly quadrilateral shape were visible through the orbital cavity in the roof anterior to

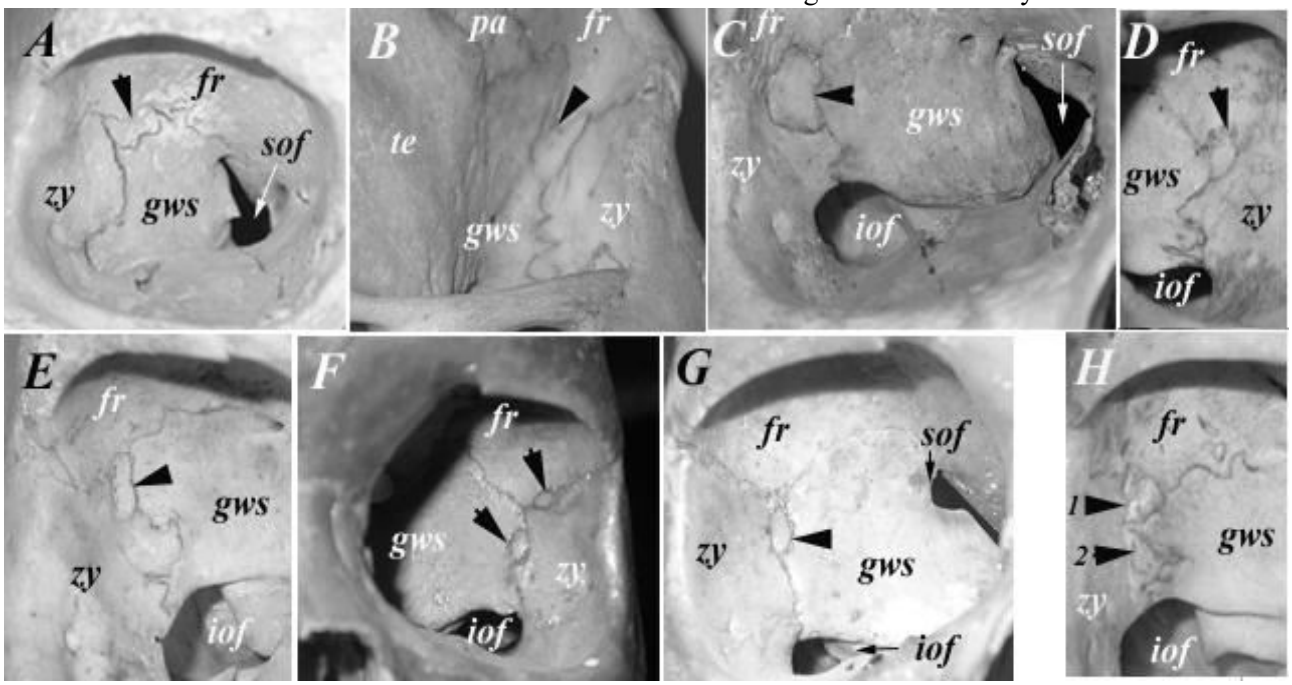

Fig. II: A-H show supernumerary bones on the lateral wall of the orbit (arrow heads) ; A- shows a large ossicle in site-1; B- same ossicle seen on the lateral aspect in the floor of the temporal fossa; C, D & E-shows the ossicles of different size and shape (site 1); F & G-shows small island of bones along spheno-zygomatic suture (Site-2); F-shows a tiny ossicle along the fronto-zygomatic suture close to the orbital opening; H-shows two ossicles one below the other with a gap in between along the spheno-zygomatic suture.

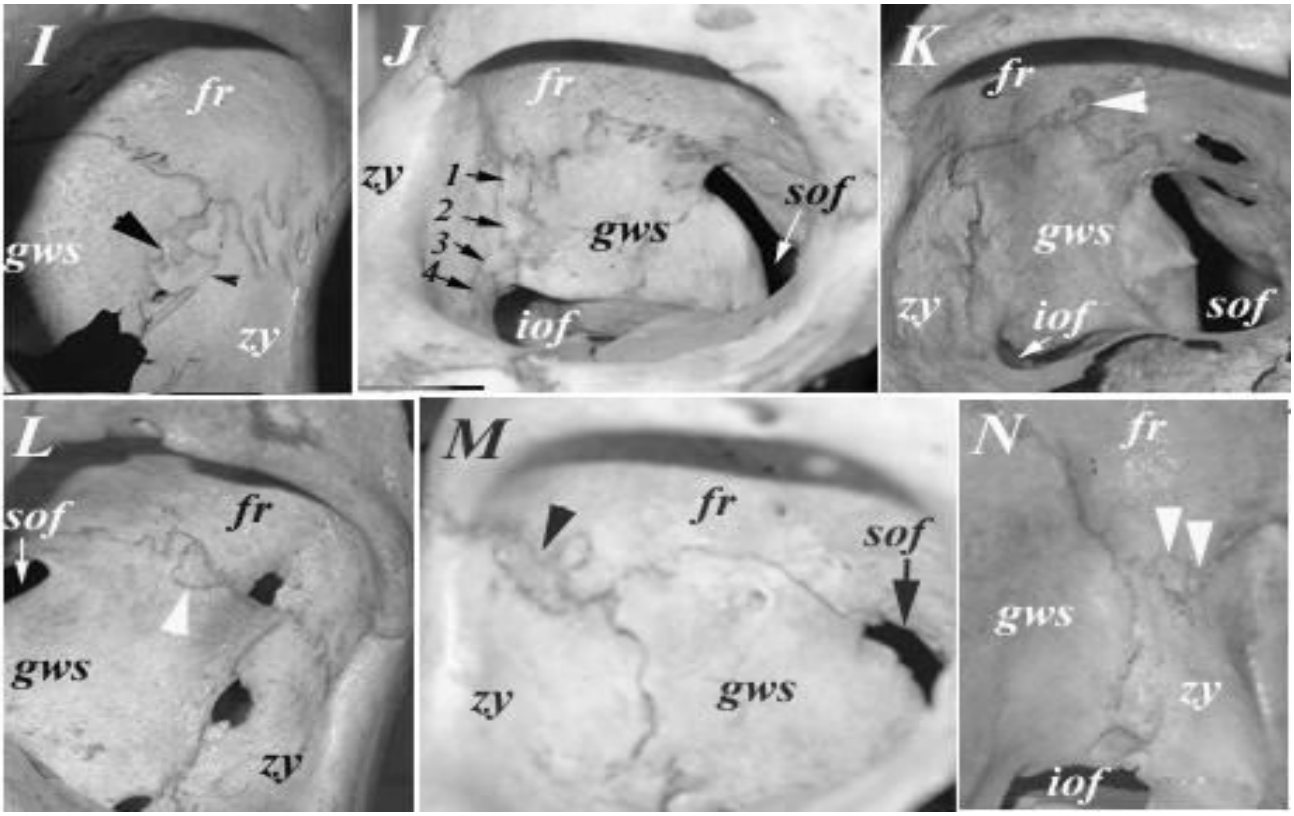

Fig. III: I-shows a cluster of ossicles grouped in the middle of the spheno-zygomatic suture (site-2); J-shows four ossicles arranged one below the other along the spheno-zygomatic suture; K & L-shows tiny ossicles along the spheno-frontal suture in the right and left orbits of the same skull; M-shows an ossicle along the fronto- zygomatic suture close to orbital aperture; N-shows two tiny ossicles along the fronto-zygomatic suture on the external surface of the lateral wall of the orbit (floor of the temporal fossa).

the superior orbital fissure. These ossicles were not visible on the floor of the anterior cranial fossa (Fig. IV-O, P).

In six skulls the supernumerary ossicles were visible from the floor of the anterior cranial fosse but since they did not extend into the full thickness of the roof they were not visible from the orbital cavity (Fig. IV-Q,W). Of these, in two skulls it was bilateral and in five skulls it was unilateral (three on right side and two on left side). These ossicles were usually of irregular shape but in one case on the right side, two ossicles were found adjacent to each other; the medial one being triangular and the lateral one quadrilateral in shape (Fig. IV-T). Usually the ossicles were located anterior to the lesser wing of the sphenoid and possessed indistinct margins. In one skull on left side the ossicle had distinct margin and was of triangular shape (Fig. IV-V). No ossicles were observed on the medial wall or the floor of the orbit in the present study.

Discussion:

A number of sutural bones have been described in relation to the bony walls of the orbit in the literature (Duke-Elder, 1964):

On the medial wall: *Ossiculum maxilla-frontale* in the lateral aspect of the frontal process of the maxilla,*os maxilla-naso lacrimale* in the medial wall of the angular process of the frontal bone, small *ethmo-lacrimal ossicles* in the ethmo-lacrimal sutures. Small wormian bones may be found behind the ethmoid and between the palatine and sphenoid bones. The hamular process of the lacrimal bone may form a separate ossicle – *the os hamulus* of Macalister, or may be separated from the orbital floor by an accessory Ossicle, the *ossiculum canalis naso-lacrimalis* of Gruber (Macalister, 1884; Le double, 1906; Gruber, 1887).

Lateral wall: Infrequently accessory ossicles are known to occur in the spheno-zygomatic suture on the lateral wall (Last, 1968; Bron et al, 1997; Black, 2008). The lateral wall is the thickest of the orbital walls. Triangular in outline, it is formed by the orbital surface of the greater wing posteriorly and orbital surface of the frontal process of the zygomatic bone anteriorly.It is traversed horizontally near the roof by the suture between the frontal bone and the upper borders of the greater wing of the sphenoid and the zygomatic bones, and vertically by the suture between the zygomatic and greater wing of the sphenoid bone. Developmentally

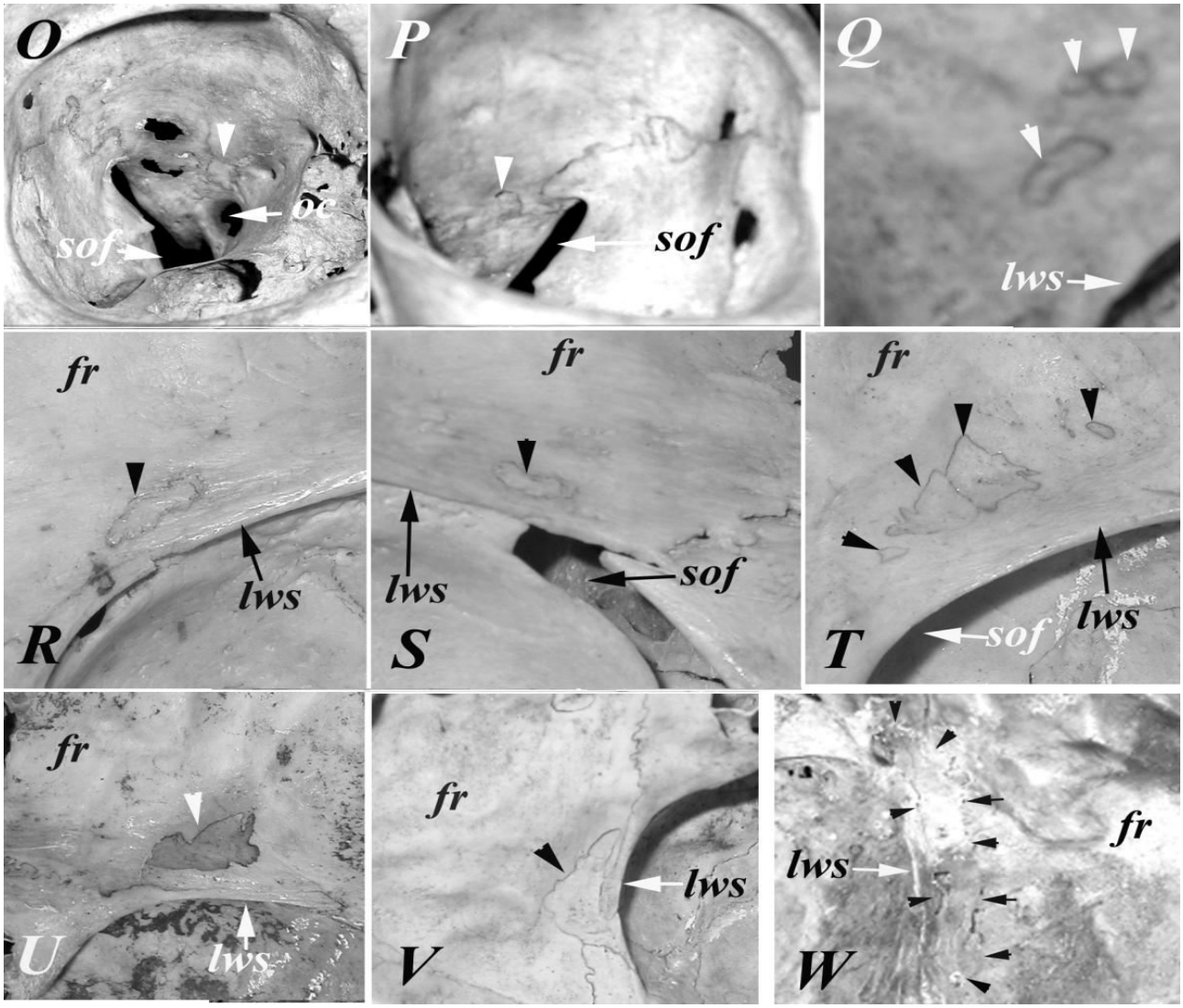

Fig. IV: Shows ossicles in the roof of the orbit. O & P-shows small ossicles in the roof of the orbit located anterior to the superior orbital fissure as seen through the right and left orbits of the same skull ; Q & W-shows ossicles in the roof of the orbit as seen on the floor of the anterior cranial fossa; Q-shows a tiny ossicle in the orbital plate of the frontal bone anterior to the lesser wing of the sphenoid; R and S-shows ossicles in the orbital plate of the frontal on the left and right side of the same skull; T-shows a pair of ossicles located adjacent to each other in the orbital plate of frontal on the right side of a skull; U, V & W-shows large irregular shaped ossicles in the anterior cranial fossa.The ossicles in U and V had well defined margins but the ossicle in the W has indistinct margins due to fusion of the sutures.

Abbreviations Used: ***fr***- orbital plate of the frontal bone; ***gws***-greater wing of the sphenoid; ***iof*** –inferior orbital fissure; ***lac***-lacrimal bone ***lws***-lesser wing of the sphenoid; ***max***- maxilla; ***oc***-optic canal; ***pa***- parietal bone, ***sof***- superior orbital fissure; ***te***- temporal bone, ***zy***- frontal process of the zygomatic bone.

both the bones forming the lateral wall are formed by membranous ossification (Inkster, 1951; Last, 1967; Black, 2008).

In the present study the sutural bones were found along the spheno-zygomatic, fronto-zygomatic sutures and at the junction where the sutures between the frontal, zygomatic and greater wing of the sphenoid meet.

The mechanism for the formation of the sutural bones is not precisely known. They have been variously linked with rapid cranial expansion , metabolic disorders of the mesoderm , head stress including pathology and hydrocephaly as the basis (Dorsey,1897; Hess, 1946; Inkster,1951; Bennett,1965; Finkel,1971). Skulls showing sutural bones in the lateral wall of the orbit in the present study did not show any evidence of cranial pathology or deformities. It is opinion of many workers that sutural bones derive from normal developmental process and are genetically determined (Finkel, 1971).

As such only few studies dealing with anomalies and pathology of the bony orbit are available which have made a brief mention of the occurrence of

Table-I: Prevalence of the sutural bones on the lateral wall and roof of the Orbit in the present study

Location	Bilateral (no. of cases)	Unilateral (R/L-no. of cases)	Total (%)	Mean dimensions of the ossicles in mm SD			
				Right		Left	
				vertical	transverse	vertical	transverse
Lateral Wall:							
1.Junction of the frontal, greater wing of the sphenoid and the zygomatic bones.	1	5 R;1L	7 (4.29)	6.96 2.49	8.54 3.47	3.0 1.41	2.5 0.71
2. Spheno – zygomatic suture.	0	5R; 2L	7(4.29)	5.25 2.05	4.37 2.2	4.0 0	3.0 0
3. Fronto -zygomatic suture.	0	3 R;0L	3 (1.84)	5.85 1.63	3.92 0.11	nil	nil
4. Spheno-frontal suture.	1	0	1(0.61)	4.68 0.078	3.38 1.31	3.85 0.95	4.53 1.9
Ossicles in the roof of the orbit.	2	3R;2L	7(4.29)	12.1 9.37	5.33 2.09	12.4 6.88	6.36 3.56

supernumerary ossicles in the bony walls of the orbit. No details regarding their frequency or ethnic variations in occurrence could be found in these studies (Duke-Elder, 1964; Last, 1968; Bergman et al, 1988; Bron et al, 1997; Black, 2008).

Only one report of occurrence of a supernumerary ossicle in the orbital wall could be found in the recent literature. These authors found the ossicle in the roof of the bony orbit in only one skull bilaterally among 1,276 skulls (0.8%) examined by them. The ossicle was an irregular bone found bilaterally between the orbital plate of the frontal and the lesser wing of the sphenoid bone measuring 12x10 mm. According to them this type of ossicle has not been described in the literature previously which they have named as 'os orbitale' (Malhotra et al, 1980).

According to Mafee et al (2005), anomalies in ossification may result in accessory sutures and supernumerary ossicles in the orbital walls.

In the present study, seven instances of ossicles resembling the 'os orbitale'were found in the roof of the orbits. Except in one skull where the ossicle was visible from the orbital cavity, in all the other instances the ossicles were found in the floor of the anterior cranial fossa only and these were not visible from the orbital cavity. The roof of the orbit is formed by the orbital plate of the frontal bone and the lesser wing of the sphenoid. The frontal bone develops from a pair of centers for ossification one each for right and left halves of the bone whereas the presphenoidal part of the sphenoid develops from six centers of ossification (Inkster, 1951). The supernumerary ossicles seen in the roof may result from the failure of fusion of one of these bones. Most of the supernumerary ossicles observed in the present study were found in the lateral wall of the orbit, and no ossicles were observed on the medial wall or the floor of the orbit.

Quite a few ossicles observed in the present study were sufficiently large; in one instance an ossicle measured 8x12 mms. An ossicle of such size is most likely to be visualized in a lateral skull X-ray. In cases with head injury it may be mistaken for a fractured fragment of the bony wall of the orbit or it may get dislodged due to impact of the injury and damage the intraocular soft tissue structures.

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Research Article

The Community Knowledge and Practices Regarding Dengue Fever in an Urban Slum Area of South India

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Abstract:

Dengue/Dengue Hemorrhagic Fever (DHF) is an emergent disease in India. The present study was carried out with the aim of assessing knowledge regarding Dengue fever among community in an urban slum area of Andhra Pradesh. This cross sectional study was undertaken in an urban slum area of Uuban Health Centre, Guntur, which is a field practice area of Department of Community Medicine, Katuri Medical College and Hospital,Guntur.

Out of 370 respondents, 291(78.65%) knew that dengue fever is transmitted by mosquito.Only 38(10.27%) persons could enumerate 3 symptoms of Dengue (fever, headache and bleeding). Regarding knowledge about breeding places only 276 (74.59%) respondents knew about breeding places of mosquitoes. Regarding the source of information on Dengue fever, 191 (51.62%) came to know about Dengue fever through television. Despite good awareness about dengue fever, adoption of the mosquito control methods was poor in the area.

Key Words: Community perception, Awareness, Source of knowledge.

Introduction:

Dengue infection is increasingly recognized as one of the world's emerging infectious diseases (Guzman & Kouri, 2002; Gubler, 2002; Halstead, 1999). About 50–100 million cases of dengue fever and 500,000 cases of Dengue Hemorrhagic Fever (DHF), resulting in around 24,000 deaths, are reported annually (Porter et al, 2005; WHO, 1997). Over half of the world's population resides in areas potentially at risk for dengue transmission, making dengue one of the most important human viral disease transmitted by arthropod vectors in terms of morbidity and mortality (Gibbons & Vaughn, 2002). Dengue/DHF is an emergent disease in India. It is endemic in some parts of the country and contributes annual outbreaks of Dengue/ DHF (Sharma et al, 2000). It mostly affect urban and peri urban areas. The geographical distribution of disease has greatly expanded and number of cases has increased dramatically in the last 10 years. In India, the risk of dengue has shown an increase in recent years due to urbanization,life style changes and deficient water management. Improper water storage practices in urban,peri-urban and rural areas lead to proliferation of mosquito breeding sites.During 2009 in India,about 15,509 cases were reported with 89 deaths.

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Dengue virus infection is endemic in Andhra Pradesh. In the year 2009, 1,190 laboratory confirmed cases of dengue fever had been reported in Andhra Pradesh with 11 deaths (Govt. of India, 2010). The present study was undertaken with the aim of assessing knowledge regarding Dengue fever among community in an urban slum area of Andhra Pradesh. Another aim was to assess, whether simple preventive measures to check and destroy the breeding sites of mosquito like checking of coolers, discarded tyers, flower pots etc. are being practiced in the community or not?

Material and Methods:

A present cross sectional study was undertaken in an urban slum area of UHC, Guntur, Andhra Pradesh which is field practice area of department of Community Medicine, Katuri Medical College and Hospital, Guntur.Out of 3 field practice area, one area Shrinavas Rao Thota was selected randomly for study.Total houses in the area were 1970. Every fifth house was selected by systemic random sampling method for collecting information. Therefore, 395 houses were selected for the study. Out of which 25 houses were locked at the time of visit. Hence, total houses visited were 370. In the selected house, an adult member in the family, who was present at the time of visit, was interviewed for collection of information using pre-tested close ended structured questionnaire. The present study was carried out from Jan 2010 to April 2010.

Results:

Out of 370 study subjects, 234 were males and 136 were females. Out of total respondents 287 (77.57 %) were from the age group of 26-40 years; 343 (92.70%) were literates (Table I). Overall 291(78.65%) respondents knew that dengue fever is transmitted by mosquito and 54 (14.59%) persons associated Dengue with flies/person to person transmission (Table II) Regarding knowledge about signs and symptoms of dengue, 214 (57.84 %) persons could enumerate one symptom (fever), 59(15.95%) persons could enumerate 2 symptoms (fever, bleeding) and 38(10.27%) persons could enumerate 3 symptoms of Dengue (fever, headache and bleeding) (Table III). Two hundred seventy six (74.59%) respondents knew about breeding places of mosquitoes. "Coolers" as the most probable breeding site (for mosquitoes) was named by 147 (39.73%) respondents followed by "cooler and tyres" by 78(21.08 %) respondents (Table IV). Regarding personal protection against mosquito bite, 274 (74.05%) respondents were relying upon Mats/coils and 63 (17.03 %) were using bed nets. Further, 33(08.92%) respondents did not give any comments (TableV). Regarding the source of information on Dengue fever, out of 370 respondents, 191 (51.62%) came to know about Dengue fever through television and/or radio followed, by 81(21.89%) to newspapers and banners, 41(11.08%) respondents came to know through friends, relatives and 57(15.41%) respondents through health workers.

Table I: Education status of respondents (n=370)

Education	Number	Percent
Illiterate	27	07.30
Higher secondary	33	08.92
Senior secondary	79	21.35
Graduate	141	38.11
Post graduate	90	24.32

Table II: Knowledge regarding transmission of dengue fever

Route of transmission	Number	Percentage
Mosquito	291	78.65
Flies	54	14.59
Don't know	25	06.76

Table III: Knowledge about signs and symptoms of dengue fever

Signs and symptoms	Number	Percentage
Fever	220	59.46
Headache	53	14.33
Fever, bleeding	59	15.95
Fever, headache and bleeding	38	10.26

Table IV: Knowledge regarding breeding places of mosquitoes (n=370)

Breeding site	Number	Percentage
Coolers	147	39.73
Coolers and tyres	78	21.08
Coolers, tyres and flower pots	17	04.59
Burrows and pits	09	02.43
Vessels/Containers	13	03.52
Coconut shells	12	03.24
Not aware	94	25.41

Table V: Use of Personal protection measures against mosquitoes

Personal protection measures	Number	Percentage
Mats/coils	274	74.05
Bed nets	63	17.03
Nothing	33	08.92

Discussion:

Overall 291(78.65%) respondents knew that dengue fever is transmitted by mosquito and 54 (14.59%) persons associated Dengue with flies/person to person transmission.A field-based study from Thailand (Swaddiwudhipong et al, 1992) also had similar findings. Gupta et al (1996) concluded that 71 and 89 percent respondents from rural and urban areas respectively from Delhi had the knowledge regarding transmission by mosquito. Regarding knowledge about signs and symptoms of dengue, 214 (57.84 %) persons could enumerate one symptom (fever), 59(15.95%) persons could enumerate 2 symptoms (fever, bleeding) and 38(10.27%) persons could enumerate 3 symptoms of Dengue (fever, headache and bleeding).Similar findings were also reported by study conducted in Kuala Kangsar (Hairi et al, 2003). Regarding knowledge about breeding places, 276 (74.59%) respondents knew about breeding places of mosquitoes. "Coolers" as the most probable breeding site was named by 147(39.73%) respondents followed by "cooler and tyres" by 78 (21.08 %) respondents. It has already been substantiated that people have good idea about the breeding places of mosquitoes (Swaddiwudhipong et al, 1992; Hairi et al, 2003). Two hundred twenty three (60.27 %) respondents were having redundant tyres, plastic pots and flower pots on rooftops or in their houses, and they accepted the fact, that they were never checking them for mosquito breeding. Out of 114(30.81%) persons having cooler in their house, 48 (42.11%) said that they never check coolers for mosquito breeding. Only 24(21.05 %) persons were correctly checking the cooler on weekly

basis. It may be concluded that though the knowledge regarding dengue is good in the general population, practice of checking coolers, tyres and flower pots is quite poor. Similar findings were also reported by study conducted in Brazil (Degallier et al, 2000). On the contrary, a study from Kuala Kangsar (Hairi et al, 2003) concludes a significant association between knowledge of dengue and attitude towards Aedes control.Regarding the source of information on Dengue fever, out of 370 respondents, 191 (51.62%) came to know about Dengue fever through television and/or radio followed, by 81 (21.89%), to newspapers and banners. Only 57 (15.41%) respondents came to know about dengue through health workers. This is in agreement with study done in Thailand (Swaddiwudhipong et al, 1992).

Conclusion and Recommendations:

Though the knowledge regarding dengue is good in the general population, adoption of the mosquito control methods was poor in the area.So Strengthening of surveillance along with health education to the community and proper training of health personnel can go long way in control of Dengue infection.

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Research Article

The Evaluation of Cord Blood Magnesium Level in Neonates of Magnesium Sulphate Treated Pre-eclamptic/eclamptic Mothers and its Clinical Correlation

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Abstract:

This case control study was carried out on 35 neonates born to pre-eclamptic/eclamptic mothers treated with magnesium sulphate and 35 neonates born to normal healthy mothers. Cases and controls were selected by inclusion-exclusion criteria irrespective of body weight and maturity of the babies. Magnesium sulphate administered to mother in study group was according to Pritchard's intramuscular regime (1984).

The cord blood samples were collected just after birth in both study and control group and magnesium level was estimated by calorimetric method. Magnesium level was found to be significantly higher in study group (6.0305±0.08047) than controls (1.916± 0.300; p = 0.0001). There was a significant difference in Apgar scores of study and control group. A negative weak linear correlation was found between cord blood magnesium level and different neonatal parameter like apgar score, birth weight and gestestional age. A weak positive linear correlation was found between magnesium level and neonatal mortality which was also not significant.

Key Words: Pre-eclampsia, Eclampsia, Magnesium sulphate.

Introduction:

Toxemia of pregnancy is a disease known from the time of Hippocrates. Pre-eclampsia is a multi-system disorder of unknown etiology characterized by hypertension of 140/90 mmHg or more with proteinuria after 20 weeks of gestation in a previously normotensive and nonproteinuric woman. Pre-eclampsia complicated by convulsion or coma is eclampsia.

Pre-eclampsia affects approximately 3% of all pregnancies worldwide. In India, the incidence of pre-eclampsia among hospital patients is about 7-10% of all antenatal admissions and that of eclampsia is about 0.94 to 1.8% the incidence in primigravidae is about 10% and in multigravidae is about 5% (Dutta & Konar, 2011).

Management of these cases has always been a controversial subject.Many drugs have been tried starting from chloroform, potassium bromide, morphine to diazepam (Duley et al, 2010), magnesium sulphate (Pritchard et al, 1984) and phenytoin (Slater et al, 1987).

When magnesium sulphate is administered parenterally to mother, it promptly crosses the placenta to achieve equilibrium with fetal serum. No untoward effect of magnesium sulphate has been noticed on babies after birth. Respiratory depression and hyporeflexia have been observed in newborns delivered by mothers undergoing intravenous magnesium sulphate therapy ,but intramuscular magnesium sulphate therapy has not been associated with any neonatal compromise. Recently a possible protective effect of magnesium has been demonstrated against cerebral palsy in very low birth weight infants (Nelson & Grether, 1995; Schendel et al, 1996, Crowther et al, 2003) .

This study was carried out to evaluate neonatal outcome in pre-eclamptic and eclamptic mothers, who received magnesium sulphate therapy intramuscularly.

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Materials and Methods:

This study was a case control study conducted in the Department of Pediatrics in active collaboration with the Department of Obstetrics & gyaenecology and Pathology, MLB Medical College, Jhansi. The period of study extended from January 2010 to September 2011.

The study was carried out on neonates born to pre-eclamptic/eclamptic mothers and normal mothers admitted in the labour room of the department of obstetrics & gyaenecology.

Neonates included in the study were divided into two groups : Group A-Study group
Group B-Control group.

Group A:

Inclusion Criteria for the study group:

Neonates born to mothers who had high blood pressure (>140/90mmHg) associated with proteinuria

and with or without convulsion or coma.

Exclusion criteria for the study group:

Babies born to mothers having following conditions were excluded from the study.

i. Eclamptic mothers with convulsions occurring after delivery or pre-eclamptic and eclamptic mothers with dead fetus *in utero*.
ii. Medical complications including heart disease, diabetes mellitus, chronic renal failure, thyrotoxicosis etc.
iii. Pre-pregnancy systemic hypertension, respiratory disease or history of epilepsy.

Selected mothers received magnesium sulphate therapy according to Pritchard's regime.

Group B:

Control group comprised of neonates born to healthy mothers who had normal BP throughout pregnancy with no proteinuria and no edema.

After selection of cases, detailed antenatal, natal and postnatal history with clinical examination was recorded in the proforma.

Following investigations were carried out:

In mother: Routine investigations including Hb, TLC, DLC, ESR, blood sugar, blood urea, urine routine / microscopy was done. Special investigations carried out in the study group were serum creatinine, serum electrolyte, liver function tests, total protein, serum albumin, serum globulin, AG ratio, urinary proteins.

In new born: Magnesium was estimated in cord blood collected at the time of delivery using magnesium kit (Calmagite method).

Observations:

In the present study, mean age of mother in the study and the control group was 24.09±4.39 and 26.62±4.80 years respectively. The difference was statistically significant ($p<0.05$). In the study group 65.7% and in control group 42.86% mothers were primigravida, the difference in parity was statistical significant.

Mean gestational age of neonates in the study group was 34.89 weeks, in contrast to 36.63 weeks in the control group. The difference was significant ($p<0.05$). The mean birth weight of newborn in the study group was 2165.7 gm against 2605.14 gm in the cotnrol group and the difference was significant ($p<0.05$). The mean apgar score in the study and in the control group was 6.45±1.83 and 8.05±1.12 respectively and the difference was highly significant ($p<0.0001$; Table I).

Mean dose of magnesium sulfate administered to mothers in the study group was 33.50±14.30 gm and mean duration of administration was 18.33±10.489 hours. Mean cord blood level of magnesium was higher in the study group as compared to the control group and the difference was highly significant ($p<0.0001$).

Neonatal mortality was higher in the study group as compared to the control group but the difference was not statistically significant ($\chi^2=2.25$, d.f=2, p=0.32). However, neonatal morbidity in terms of prematurity ($\chi^2=4.632$, d.f=1, p=0.03), low birth weight ($\chi^2=5.733$, d.f=1, p=0.01), asphyxia ($\chi^2=4.590$, d.f=1, p=0.032), IUGR ($\chi^2=4.928$, d.f.=1, p=0.02) was significantly higher in the study group as compared to the control group (Table II).

In the present study, it was observed that there was insignificant weak negative correlation between cord blood magnesium and neonatal parameters like apgar score, birth weight and gestational age ($p>0.05$). There was a weak positive linear correlation between magnesium level and neonatal mortality which was also not significant ($p>0.05$).

Table I: Comparison of different parameters between the study and control groups.

Parameter	Study Group Mean ±SD (n=35)	Control Group Mean ±SD (n=35)	*z*-value	*p*-value
Age of mother (in years)	24.09 ± 4.39	26.62 ± 4.80	2.298	0.0215*
Gestational age (in weeks)	34.89 ± 2.84	36.63 ± 2.45	2.740	0.006 *
Birth weight (in grams)	2165.7 ± 642.97	2605.14 ± 538.32	-3.103	0.0019*
Apgar score	6.45 ± 1.83	8.05 ± 1.12	18.161	0.0001**
Cord blood magnesium level (in mg)	6.0305 ± 0.08	1.916 ± 0.30	28.3273	0.0001**

*Significant ($p<0.05$), ** Highly Significant ($p<0.0001$)

Table II: Bivariate correlation analysis with their significance (2 tailed) between cord blood magnesium level and different neonatal parameters in study group.

		Apgar Score	Birth Weight	Gestational Age	Neonatal Mortality
Cord blood	Correlation *(r)*	-0.228	-0.234	-0.174	+0.051
Magnesium level	Significance *(p)*	0.1869	0.1747	0.2353	0.7748

Discussion:

Toxemia of pregnancy is one of the leading cause of maternal and perinatal mortality and morbidity not only in developing countries but also in most developed nations (Lopez-Liera et al, 1976).

Newton (1964) found the incidence of toxemia in primigravida to be 70%; Pritchard et al (1984) found this incidence to be 85%, as against 65.7% in the study group of the present study.

In the present study, 65.7% neonates of the study group were, below 37 weeks of gestation, similarly Mohanty et al (1990) found 66% cases to be below 37 weeks of gastation. In the present study significant difference was observed in birth weight in both the groups. Twenty four out of 35 babies of the study group and 13 out of 35 babies of the control group were of low birth weight in the present study.

At 1 minute, 91.2 % babies had apgar score of <7 and 39.9% babies had apgar score of <7 at the end of 5 minutes in the study group while in the control group, 48.55% babies had apgar score of <7 at the end of 1minute and only 8.5% babies had apgar of <7 at the end of 5minutes.

Crowther et al (2003) reported 44% babies to have apgar score of <7 at 1 minute and in 22% apgar score of <7 at 5 minutes in a group of toxemic mothers treated with magnesium sulphate.

Study conducted by eclampsia collaborative group (1995) observed that 48.9% neonates had apgar score of <7 at 1 minute while apgar score at the end of 5 minutes was of <7 in 22.3% neonates.

Eighty percent babies in study group survived beyond 72 hours, whereas 91.4% newborns survived beyond 72 hours in the control group. Although neonatal mortality in study group was higher, but the difference between the two groups was not statistically significant.

In the present study, the perinatal mortality in study group was 17.05%, however, Crowther et al (2003) reported the perinatal mortality to be 7.4%; Mohanty et al (1990) reported perinatal mortality to be 31%, and in a randomized control trial by Magpie trial collaborative group (Altman et al, 2002) found it to be 12.7%.

In the present study, cord blood magnesium level was higher in neonates in the study group (6.03±0.80 mg/dl) as compared to neonates in the control group (1.91±0.30 mg/dl).

The above observations were in accordance with the study of Cruikshank et al (1979), who suggested that at the time of delivery, the offsprings of the $MgSO_4$ exposed women were hypermagnesemic, although less so than their mothers. Mason et al (1996) found that total magnesium and ionized magnesium were significantly ($p<0.001$) elevated in the cord blood samples of the treated group. Perveen et al (2002) suggested that magnesium may reach the fetus and appear in the cord blood at a higher than normal concentrations.

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Research Article

Efficacy of Yoga and Swimming in Reducing Anxiety: A Comparative Study

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Abstract:

Different exercise modules and yogic practices have been claimed to reduce anxiety. However, there are very few longitudinal studies to assess and to compare improvement in mental health of subjects performing yogic asanas and breathing exercises versus those performing endurance exercises like swimming. Therefore, present study was designed to compare reduction in anxiety levels with yogic postures and breathing exercises with that of swimming.

This study was conducted in the Department of Physiology. Study design used was prospective randomized comparative Study. Hundred volunteers were included in the study and randomly divided into two groups; one practiced yogic asanas and breathing exercises and other practised swimming for 12 weeks. Beck's Anxiety Inventory was used to assess anxiety level of subjects. Anxiety levels were assessed prior to the training and then after 12 weeks of training. The total score was calculated from 21 items and high scores indicated higher anxiety levels. The average anxiety scores decreased significantly ($p<0.0001$) in both the groups after training. In yoga group. average pretraining score of 24.25 decreased to post training score of 20.27, whereas in swimmer group it decreased from 23.57 to 20.8. However, the decrease in anxiety was similar with both modalities of exercise ($p>0.05$).

Key Words: Anxiety, Exercise module, Swimming, Yoga.

Introduction:

Anxiety is pathological when excessive and persistent, or when it no longer serves to signal danger. It is often considered to be a major component of unhealthy lifestyle and possibly contributes significantly to the pathogenesis of not only psychiatric but also systemic disorders such as cardiovascular disease, diabetes mellitus and bronchial asthma (Gupta et al, 2006).

The vast majority of studies have shown decrease in tension and depression with acute bouts of moderate intensity exercise (Berger & Motl, 2000). Moderate intensity exercise might not optimize fitness and sport training benefits, but it had consistently been associated with desirable mood changes (Berger & Owen, 1988). Many studies have proved efficacy of yoga in reducing anxiety (Gupta et al, 2006; Brown & Gerbarg, 2005).

However, randomized prospective comparative studies between effects of endurance exercises and yoga (postures and pranayama) on reduction in anxiety are few. Therefore, the present study was undertaken to evaluate and compare the efficacy of swimming and yoga as modules (1 hour daily, 6 days a week) for reducing anxiety.

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Material and Methods:

Study Set Up:

The study was conducted in the Department of Physiology in Post Graduate Research Laboratory, Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha in co-ordination with "Anekant Swadhyaya Mandir (Yoga Centre)", Ramnagar, Wardha and "Municipal Swimming Pool", managed by Police Welfare Fund, Civil Lines, Wardha.

Study Groups:

Healthy males and females with normal physical examination and with sedentary occupations between 18 and 40 years of age were included in the study. The volunteers from the general populations were motivated to participate in the study by explaining plan of the study to them. The subjects were included purely on the voluntary basis. After screening and fulfilments of inclusion and exclusion criteria, volunteers were included in the study. Initially 100 volunteers were recruited but at the end of the study, Yoga group consisted of 41 subjects (n=41) out of which 16 were males and 25 females. Swimmer group comprised of 40 subjects with 18 males and 22 females. Volunteers had not been engaged in yoga practice or swimming nor were they doing any physical exercise at least during 3 years preceding the study. Subjects in non-sedentary occupations, smokers, alcoholics, pregnant female, postoperative patients and subjects suffering from any hernia, cardiovascular disorder, any active respiratory

tract infection or history of respiratory tract infection during previous 6 weeks, were excluded from the study by detailed history and thorough clinical examination.

Study Protocol:

Clearance of Institutional Ethics Committee was obtained. After selection of the subjects, they were explained about the detailed plan of work and aim of the present research project. A written informed consent was obtained from them.

One hundred volunteers were divided into cohorts of 10 subjects each and were randomly assigned (block randomization) to undergo either yogic training or swimming for a duration of 12 weeks. Before the actual training period, baseline parameters were recorded in a week's time for one cohort. In the same week the subjects of that cohort were motivated for the exercise regimen they had to follow during the entire 12 weeks period. After 12 weeks exercise by all ten subjects in that cohort, all the parameters were studied again. After baseline parameters were recorded for one cohort and the training started for that cohort, the next cohort was subjected to the same treatment. Out of the 100 subjects, 9 from the yoga group and 10 from the swimmer group dropped out in due course of the study. Thus, at the end of the study, data of 41 subjects from yoga group and 40 subjects from swimming group were analyzed.

The subjects of yoga group were instructed not to practice any yogic technique other than the prescribed ones and swimmer group was advised to refrain from other physical exercises during the study. We supervised the subjects early in the morning (5.00 - 6.00 a.m) during yoga classes and swimmers from 6.00 – 7.00 a.m. every day during the training period. Participants of both the groups were allowed to do their routine activities during the study period.

The subjects were taught yogasanas and pranayamas and then they practised the same, 6 days/ week for 60 minutes daily, for a total duration of 12 weeks. Iyengar yoga techniques were followed by the yoga trainers (Iyengar, 1995). Different yogasanas (yogic postures) viz. tâdâsana, konâsana, utkatâsana, sarvângâsana, halâsana, chakrâsana, padmâsana, dhanurâsana, makarâsana, pashchimottânâsana, vajrâsana, virâsana and shavâsana were practiced for 40 minutes and pranayamic breathing exercises with purak, rechak and kumbhak, anulom-vilom, bhastrikâ, bhramari prânâyâm and kapalbhâti were practised for 20 minutes. Swimming was practiced 6 days/week for 60 minutes daily. Swimming comprised freestyle in first 6 weeks (including training in first 2-3 weeks) and freestyle and breast stroke in last 6 weeks including 10 minutes of floating on the water. For novice swimmers, continuous swimming for 60 minutes is difficult; therefore, intermittent floating with deep slow breathing was introduced. It also helped to keep similarity with yoga group who practiced shavâsana for 10 minutes (Lying still and relaxed with slow deep breathing). An important limitation of the methodology was inability to compare the intensities of two modalities of exercise during 12 weeks duration. This inability was because of the fact that unlike endurance exercise, intensity of exercise for yogic asanas and pranayama is not directly related with exercise and post-exercise heart rates.

Measurement of Anxiety:

Beck's Anxiety Inventory (BAI) was used for the assessment of anxiety levels of subjects of both the exercise groups. Beck's Anxiety Inventory scale has good reliability and validity with high internal consistency and item-total correlations ranging from .30 to .71 (median=.60) and the correlations of the BAI with a set of self-report and clinician-rated scales are also all significant. Each subject was properly explained the procedure for filling the inventory; they filled the inventory during initial visit for basal parameters recordings prior to the training and post-training parameters after 12 weeks of training.

The inventory consisted of 21 questions, which the subjects were asked to fill within 10- 20 minutes. Against each question; columns labelled 0,1,2,3 were printed and subjects were required to mark (√) in the column which they felt appropriate.

The total score of each column helped in calculating the grand total of all 3 columns. The total score for all 21 questions was calculated. High scores indicated higher anxiety levels. Maximum grand score attainable was 63. A grand total between 0-21 indicated very low anxiety, between 22 and 35 indicated moderate anxiety and a score that exceeded 36 was considered a potential cause for concern (Beck et al, 1988).

Statistical Analysis:

All the data obtained was presented group-wise by descriptive statistics using mean, and standard error of mean. For differences in sex-wise composition of two study groups, Chi squared test was used. For each parameter in both yoga and swimming groups before and after training period of 12 weeks, data

distribution was tested for normality of distribution by Kolmogorov Smirnov test. As the data distribution was not normal, data were log converted and again tested for normality of distribution.

As the data distribution (Anxiety scores and log converted data) was not normal, the paired data before and after the exercise for both yoga and swimming groups was tested by Wilcoxon signed rank test.

The change in anxiety scores with exercise was studied by calculating delta i.e. difference in value before and after the exercise of both modalities. The percent change was also calculated for each parameter as percentage of change with respect to pre exercise level score. Percentage increase or decrease in value of a parameter (delta) with yoga and swimming was also compared using Mann Whitney U test as data distribution was not normal.

The statistical significance was considered at probability value less than 0.05.

The statistical calculations were done using Data Analysis tool of Microsoft Excel and Systat 12 (Systat Software, Inc. Chicago).

Results:

Yoga and swimming groups were statistically comparable with respect to age and sex distribution as shown in Table I. The average anxiety scores decreased significantly with both modalities of exercise after 12 weeks training (Wilcoxon signed rank test, $p<0.0001$). The decrease in average anxiety scores were similar in with both yoga and swimming (Mann Whitney U test, $p>0.05$) as shown Table III.

Table I: Comparison of the yoga and swimming groups

	Yoga (n=41)	Swimming (n=40)	Statistical significance
Age (years)	28.439	27.7	$t = 0.7338$, df = 79
(Mean ±S.E.)	± 1.41	± 1.35	$p = 0.7067$
Males (n)	16 (39.0%)	18 (45%)	$\chi^2 = 0.2968$, df = 1
Females (n)	25 (61.0%)	22 (55%)	p=0.5858

Discussion:

Decrease in anxiety scores were observed following 12 weeks of yoga as well as swimming with almost similar effects with both yoga and swimming though percent improvement in anxiety scores was slightly better with yoga. Thus the hypothesis that a change in anxiety would be different with yoga and swimming was not supported.

Table II: Effects of exercise on Anxiety scores of Beck's Anxiety Inventory in yoga and swimming groups

Parameter	Exercise Modality	Baseline before exercise (Mean ± SEM)	After 12 weeks (Mean ± SEM)	Wilcoxon signed rank test
Anxiety score (Beck's)	Yoga (n=41)	24.25 ± 0.936	20.275 ± 1.084	z=5.19 p<0.0001**
	Swimming (n=40)	23.575 ± 1.062	20.8 ± 1.014	z=5.3 p<0.0001**

** Highly Significant ($p<0.0001$)

Table III: Comparison of effects of exercise on Anxiety scores of Beck's Anxiety Inventory in yoga and swimming groups

Parameter	Exercise Modality	Change in anxiety scores after training (Percentage)	Mann-Whitney U-test
Anxiety score (Beck's)	Yoga (n=41)	-3.975 (-16.39%)	z=-1.73 p=0.0836
	Swimming (n=40)	-2.775 (-11.77%)	

The vast majority of studies have shown decrease in tension, depression, anger, and confusion associated with acute bouts of moderate intensity exercise. Moderate intensity exercise may not optimize fitness and sport training benefits, but it has consistently been associated with desirable mood changes (Berger & Owen 1988; Cox et al, 2001).

Short term mood improvements have also been reported after the yoga classes. Yoga produces many beneficial emotional, psychological and biological effects and it may be easy to implement (Shapiro et al, 2007; Patel & North, 1975). Shavasan, a yogic relaxation posture, has been reported to control psychophysiological stress (Patel & North, 1975; Bera et al, 1998).

There is an altered autonomic homeostasis in response to real life stressors with a shift towards cardiac sympathetic activation and vagal withdrawal. This shift towards sympathetic may be the reason of anxiety (Srinivasan et al, 2006). Cardiac autonomic modulation at rest in subjects engaged in regular exercise goes in parasympathetic favour with substantial increase in high frequency (HF) component of heart rate variability and reduced Low frequency/ High frequency ratio (Sandercock et al, 2005). Decrease in sympathetic activity has also been reported

in subjects doing yoga practice (Vempati & Telles, 2002). This favourable autonomic modulation in subjects engaged in physical activity may be responsible for reduced response to stress in the form of reduced anxiety.

With limitation of measuring intensity of exercise in yoga for comparison with swimming (as heart rates in yoga postures and pranayams are not intensity related), the evaluation of swimming and yoga as modules for reducing anxiety was done in the present study. However, the time for the intervention in both modes was same (1 hour daily, 6 days a week for 12 weeks). We found similar reduction of anxiety by these two modules of exercises though the results of the present study needs further confirmation on a larger sample size as adequacy of sample size was not tested in the present study. Similarly inclusion of only randomly selected subjects in such type of longitudinal study from general population is difficult (though included subjects were randomly assigned to two modalities of exercise) and, therefore, generalization of the results of present study must take into account this limitation.

Our finding that reduction in anxiety is similar with swimming and yogic exercise module is consistent with the finding of Cox (2000) who have reported that change in anxiety is not associated with mode of exercise. However, Berger & Owen (1988) and Steptoe & Cox (1988) have proposed that modes that are more aerobic, noncompetitive, more predictable, and repetitive are believed to promote a greater degree of stress reduction. While acute bouts of aerobic exercise are associated with immediate or delayed anxiolytic effects, resistance exercises such as recreational weight lifting does not seem to reduce the anxiety (Raglin et al, 1993; Koltyn et al, 1995). Sime (1977) has reported that the physiological response to stressors in reducing heart rate and electrodermal response is better with exercise than with meditation. Bahrke & Morgan (1978) have found that acute physical activity, noncultic meditation, and a quiet rest session are equally effective in reducing state and trait anxiety.

In conclusion, yoga (yogic postures and pranayama) as well as swimming significantly reduce anxiety within 12 weeks. However, reduction in anxiety is similar with both yoga and swimming. Therefore, both yoga and swimming can be advocated for alleviation of anxiety. In addition, other factors like cost effectiveness, facilities for recreational exercise, physical constraint, training facilities and ability of any exercise regime to keep continued motivation and interest of the trainees should also be taken into account for exercise prescription.

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Research Article

Study of Association of Serum Lipids with Diabetic Retinopathy in Type 2 Diabetes Mellitus

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Abstract:

The present study was conducted to find out the role of serum lipids in the development of diabetic retinopathy in type II Diabetes Mellitus. One hundred fifty subjects aged 30-70 years attending OPD at Old Civil hospital, Surat, participated in the study and were divided into three groups. Group I included 50 healthy non-diabetic subjects who served as control. Group II included 50 diabetic subjects with no signs of diabetic retinopathy and Group III included 50 diabetics with diabetic retinopathy. Funduscopy under homatropine was done in all the subjects. Serum triglycerides and total cholesterol were estimated by enzymatic methods and High Density Lipoprotein by precipitation method. Serum Low density lipoprotein was calculated using Friedewald's formula. It was found that triglyceride levels were significantly raised ($p<0.05$) in subjects with diabetic retinopathy as compared to those without diabetic retinopathy showing a positive association of Triglycerides with the incidence of diabetic retinopathy. Whereas no such association was found between low density lipoprotein and total cholesterol levels with the prevalence of diabetic retinopathy.

Key Words: Triglycerides (TG), Total Cholesterol (TC), Low Density Lipoproteins(LDL), Diabetic Retinopathy(DR).

Introduction:

In 2009-10 out of 285 million people suffering from Type II Diabetes Mellitus (DM) worldwide, 51 millions were Indians as per International Diabetic Federation. Diabetes along with its fatal complications, is one of the leading cause of mortality and morbidity. Chronic complications of DM includes macro vascular complications like coronary artery disease, cerebrovascular disease and peripheral vascular disease along with microvascular complications like retinopathy, nephropathy and neuropathy. Risk factors like duration of diabetes, glycemic control (HbA1c), systolic blood pressure, dyslipedemias, smoking and microalbuminurias have been linked with complications of DM. Various studies have shown a positive correlation between elevated serum lipids (TG, LDL, TC) and macrovascular complications like ischemic heart disease. However, studies of association of elevated serum lipids with microvascular complications like diabetic retinopathy (DR) have shown varying results. In this study attempt has been made to quantify and specify the role of various components of serum lipids with the prevalence of DR.

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Material and Methods:

The present study was carried out on 150 subjects aged between 30-70 years attending the outpatient department at Old Civil Hospital, Surat. A written consent was taken from all subjects and details of procedure were explained to them in the local language. A detailed medical history and findings of clinical examination were recorded in a proforma. Subjects with type-I DM, type-II DM of less than 5 years duration, severe hypertension, acute infections, known cardiovascular and renal diseases, liver dysfunction, severe anemia and thyroid disorders were excluded on the basis of history, examination and routine investigations like blood test, chest X-ray, electrocardiogram (ECG) and urine analysis. After overnight fasting and 2 hours after meals, fasting and postprandial blood samples were obtained from all the subjects. Fasting Blood Sugar (FBS) and Postprandial Blood Sugar (PPBS) were estimated by Glucose oxidase-peroxidase method. Funduscopy was done after dilatation using homatropine. Subjects with signs of micro-aneurysm, retinal dot blot hemorrhages, cotton wool spots, hard exudates is Non progressive Diabetic Retinopathy and neovascularisation progressive Diabetic Retinopathy were labeled as diabetic retinopathy. The 150 subjects were divided into three groups as follows:

Group I: 50 subjects with FBS ≤110mg% and

PPBS ≤ 140mg% without any drug or insulin, were taken as control group. GroupII: 50 subjects with history of DM for more than 5 years but no signs of DR.
Group III: 50 subjects with DM for more than 5 years and signs of DR.

Serum lipids were measured in the fasting blood sample. Serum triglycerides (TG) and cholesterol were measured by enzymatic methods, Glycerol 3 phosphate oxidase N-ethyl sulphopropyl anisidine and cholesterol oxidase-peroxidase end point methods respectively. Serum high density lipoprotein (HDL) was estimated by precipitation method Polyethylene glycol precipitation method. Serum low density lipoprotein (LDL) was calculated using Friedwald's formula (LDL=TC-HDL-TG/5). The data obtained was compared statistically by applying oneway anova test.

Results:

Seventy eight percent subjects in Group II and 92% in Group III were more than 45 years of age as compared to 66% in the control group. Retinopathy is a rare finding (8%) in subjects of less than 45 years of age (Table I). This suggests that Type II DM is common after 4th decade of life and the risk of DR also increases with the increase in age; it may be due to increased duration of the disease.

Table I: Age wise distribution of the subjects participating in the study.

Age in years	Group I	Group II	Group III
30-44	34%	22%	8%
45-59	48%	62%	46%
60-75	18%	16%	46%

Seventy two percent in Group II and 68% in Group III were males as compared to 64% males in the control group. Results show that with increase in the duration of disease there was an increase incidence of DR ($p<0.0001$). Fasting blood sugar and HbA1c were significantly raised in those with DR as compared to those without DR ($p<0.05$), both of which are significantly raised as compared to the control group ($p<0.0001$).This suggests the role of poor glycemic control (raised HbA1c) in the prevalence of diabetic retinopathy (Table II).

A raised levels of TG, LDL and Cholesterol was observed in the Diabetic subjects (Group II and III) as compared to the control Group which was highly significant ($p<0.0001$). On comparing Group II and III, it was observed that TG levels were significantly raised ($p<0.05$) in those with DR as compared to those without DR, where as LDL and cholesterol were not found to be significantly raised ($p=0.22$) in Group III as compared to Group II. This suggest that TG is associated with the increased incidence of diabetic retinopathy in DM subjects and not LDL or cholesterol.

Table II: Comparison of sex, duration, FBS and HbA1c levels in the three groups.

Variables	Mean+SD			*f*-value
	Group I	Group II	Group III	*p*-value
Sex	32males & 18 females	36 males & 14 females	34 males & 16 females	-
Duration of DM (in years)	-	7.6±2.2	10.2±3.4	-
FBS (mg %)	78.08±12.12	128.64±19.5	136±22.8	142.43 <0.0001**
HbA1c (%)	5.67±0.35	7.88±0.77	9.92±1.5	227.43 <0.0001**

Table III: Comparison of serum lipids in the three groups.

Variables	Mean+SD			*f*-value
	GROUP I	GROUP II	GROUP III	*p*-value
Cholesterol (mg%)	165.9±23.19	200.74±38.74	212.35±54.05	17.67 <0.0001**
HDL (mg%)	48±12.69	43.15±7.69	42.54±11.6	3.78 <0.025*
LDL (mg%)	106.39±30.16	127.61±34.8	136 .21±34.8	10.60 <0.0001**
TG (mg %)	133.74±38.74	150.57±44.74	168.28±40.26	8.73 <0.0001**

*Significant ($p<0.05$), ** Highly Significant ($p<0.0001$)

Discussion:

Various studies have proven the role of elevated serum lipids with macro vascular complications of DM like coronary artery disease but, studies of association of lipids with specific micro vascular complications of DM like retinopathy have shown varying results**.** Dornan et al (1982) first showed the an association of LDL cholesterol with diabetic retinopathy. In Wisconsin epidemiological study of diabetic retinopathy, Klein et al (1999) correlated raised cholesterol levels with macular hard exudates. Early treatment diabetic retinopathy study associated TC and LDL with the onset and severity of retinal hard exudates (Chew et al, 1966). Severity of retionphaty was positively associated with TG in type I DM and negatively associated with HDL choestrol in DCCT/ EDIC cohort (Lysons et al, 2004). Mohan et al (1984) reported an association between raised LDL and macular edema in the Indian population. Later Rema

et al (2006) showed association of TG with DR and LDL with diabetic macular oedema in Chennai Urban Rural Epidemiology Study Eye study (CURES) 2. However, Larsson et al (1999) and Hove et al (2004) found no association between TG, TC and HDL with diabetic retinopathy. Kulshreshtha et al (1979) observed raised levels of cholesterol and NEFA (non esterified fatty acids) in patients of DR. Benarous et al (2011) reported that phospholipids are not associated with DR but with Clinically significant macular oedema. Sasongko et al (2011) showed that apolipoprotein AI is inversely related and apolipoprotein B is directly related to DR and are strong biomarkers of DR than lipid profile in Australian population. Ozer et al (2009) found no correlation between serum lipids and macular edema in diabetic patients. Sachdev & Sahni (2010) proved that cholesterol and LDL are risk factors for retinal hard exudates in Type II DM in North Indian population. Keech et al (2007) showed that lipid lowering agent like finofibrate, decreases the progression of DR. Uçgun et al (2007) showed that TC and LDL are elevated in patients with macular edema and hard exudates.

In the present study, it was found that TC, LDL and TG levels were significantly higher ($p<0.0001$) in diabetic subjects (Group II and III) as compared to the control group. This is due to the increased flow of glucose and fatty acids to liver due to lack of insulin. Decreased clearance of LDL and TG is due to over production of apolipoprotein B and low lipoprotein lipase activity. On comparing Group II and III it was found that TG levels were significantly raised ($p<0.05$) in patients of DR. The raised TG levels leads to increased blood viscosity and altered fibrinolytic activity which leads to formation of hard exudates. Also, TG incorporates into the cell membrane, altering its fluidity and permeability which leads to hemorrhage and oedema. This also leads to endothelial cell dysfunction and local inflammatory response releasing cytokines and growth factors which are responsible for neovascularisation in retina (Joussen et al, 2001). In the present study TC and LDL levels were not found to be significantly raised ($p>0.05$) in those with DR as compared to those without DR. The study suggest the role of raised TG and not LDL and TC in the incidence of retinopathy in Type II DM.

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Research Article

Usefulness of Serum Prolactin in Differentiating Epileptic and Pseudoseizures in Children

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Abstract:

Transient hyperprolectinemia has been reported to follow unprovoked seizures. This study was conducted in 90 children aged 1-18 years of age. The study comprised of four groups: Group-1 consisted of children with epilepsy which was further subdivided into GTCS, CPS and SPS. Group-2 comprised of children having febrile convulsions. Group-3 comprised of children suffering from non-epileptic paroxysmal events like breath holding spell, syncope and pseudoseizures or conversion reaction. Group-4 consisted of children who served as controls. Blood sample was collected within two hours of the event in all the groups. The exact interval between the event and the collection of blood sample was noted. Serum prolactin level was estimated by ELISA technique. In the present study, significant elevation of serum prolactin level was observed only in the Group-1 (28.77±15.49ng/ml) as compared to controls (9.53±2.45ng/ml) and the highest levels were observed in children with GTCS. Maximum elevation of prolactin was seen within 15 to 30 minutes post ictally. As the prolactin levels become normal after two hours of post ictal period, the test looses its significance.

Key Words: Serum Prolactin, Seizure.

Introduction:

Prolactin is secreted from the anterior pituitary gland and is inhibited by tubero-infundibular dopamine neurons in the arcuate nucleus of the hypothalamus. Abnormal electrical discharge passing through the hypothalamus may disrupt the normal functioning. Generalized neuronal discharge of a seizure stimulates the hypothalamus either directly through specific neurotransmitter changes (decrease in GABA and dopaminergic system) or through the release of other substances, thereby, causing increase in serum prolactin level during epileptic form of seizures. Acute changes in serum prolactin levels which occurred following some of the seizures may be useful in differentiating epileptic seizures from non epileptic seizures.

This study was undertaken to know whether elevated serum prolactin levels following a seizure may be used to differentiate epileptic seizure from other paroxysmal disorders of childhood.

Material and Methods:

The present study was conducted on 90 children between 1-18 years of age. They were divided into four groups after taking detailed history and their examination.

Group I included children with epilepsy. These children had an provoked seizure at the time of admission. This group was further subdivided into patients with Generalized Tonic Clonic Seizure (GTCS), Complex Partial Seizures (CPS) and Simple Partial Seizures (SPS).

Group II comprised of children with typical febrile seizures. Children with doubtful diagnostic features of febrile seizures,developmental or neurological abnormalities,prolonged seizures of more than 10 minutes and focal seizures were excluded.

Group III comprised of children with non epileptic paroxysmal events e.g. breath holding spell, syncope, night terror, pseudoseizures etc.

Group IV (control) comprised of children admitted for reasons other than fever or seizure. They were free from any known metabolic or endocrine disease and in whom the exclusion criteria were not applicable.

Exclusion criteria: Children with any metabolic disturbance, infective central nervous pathology, developmental, structural or neurological abnormalities, or patients on drugs known to alter prolactin levels (like phenothizine, haloperidol, metoclopramide, opiates, cimetidine, imipramines, fluoxetine, verapamil) were not included in the study.

Blood sample was collected within two hours of the event in all the groups and the exact interval between the event and collection of blood was noted. One milliliter of blood was taken & serum was separated by centrifugation at room temperature and

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tested withcommercially available kits (UBI Magiwel prolactin quantitative, HP-201 kit) using ELISA technique which provides quantitative estimation of prolactin in serum. In children, the normal values are less than 15ng/ml. Neonatal prolactin concentrations are high, but fall to adult levels by three months of age. Levels were considered high if values were greater than 23 ng/ml or two times more than the base line value.

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented as Mean ± SD (Min-Max). Significance is assessed at 5 % level of significance. Student unpaired *t-test* was used to find the significance of study parameters on continuous scale between two groups.

Results:

The present study was undertaken to find out the usefulness of serum prolactin in distinguishing the true epileptic seizures from pseudoseizures and other non epileptic paroxymal events.

There were 30 children (17 males and 13 females) in Group I and 20 cases each in group II, III and IV (Table I).

Group I contained 15 cases of GTCS, 8 cases of CPS and 7 cases of SPS. Group II had 20 cases of Febrile convulsions. Children with non epileptic paroxysmal events (Group III) consisted of 7 children with breath holding spell, 5 with syncope and 8 cases had pseudoseizures.

Group I had highly significant increase in prolactin levels after seizure episode than the control group ($p<0.0001$). Rise in serum prolactin level was not significant in group II & III as compared to group IV (Table II).

The mean serum prolactin level in children with GTCS was 35.06 ± 14.26 ng/ml, with range of 11.80-56.45 ng/ml; in CPS, the values were 28.3 ±12.94 ng/ml with the range of 8.45-48.50ng/ml, while in SPS the mean value was 13.74±6.92ng/ml in the range of 7.50-26.75ng/ml. On statistical analysis, the levels were found to be significantly higher in children with GTCS ($p<0.0001$). The levels were also elevated in CPS but were not significant. In SPS the levels were significantly increased than the controls ($p<0.05$; Table III).

Table I: Distribution of cases according to sex in all the groups.

Group	No. of Cases	Mean ± SD	Male	Female
I	30	8.7± 3.5	17	13
II	20	3.21± 0.7	12	8
II	20	7.87± 3.1	11	9
IV	20	7.21± 2.1	10	10

Table II: Serum prolactin level in different groups

Variables	Mean±SD Range	No. of Cases	*t*-value	*d.f.*	*p*-value
Group I	28.77±15.49	30			
	7.5-56.75		5.49	48	<0.0001**
Group IV	9.53±2.45	20			
	5.75-17.45				
Group II	11.25±3.14	20			
	6.75-17.45		1.93	38	0.0609
Group IV	9.53±2.45	20			
	5.75-17.45				
Group III	8.74±2.64	20			
	5.45-14.25		0.98	38	0.3328
Group IV	9.53±2.45	20			
	5.75-17.45				

Table III: Comparison of serum prolactin levels in subdivisions of Group I.

Variables	Mean±SD Range	No. of Cases	*t*-value	*d.f.*	*p*-value
GTCS	35.06 ± 14.26	15			
	11.80-56.45		7.89	33	<0.0001**
Control	9.53 ± 2.45	20			
	5.75-13.75				
CPS	28.73 ± 12.94	8			
	8.45-48.50		0.78	26	0.4409
Control	9.53 ± 2.45	20			
	5.75-13.75				
SPS	13.74 ± 6.92	7			
	7.50-26.75		2.39	25	0.0246*
Control	9.53 ± 2.45	20			
	5.75-13.75				

*Significant ($p<0.005$), **Highly significant ($p<0.0001$)

Serum prolactin level was found to be significantly elevated after epileptic attacks. Elevated levels were observed in 12 out of 15 (80.0%) cases of GTCS, 5 out of 8 (62.50%) cases of CPS and 2 out of 7 (28.5%) cases of SPS.

Elevation in serum prolactin level after the seizure was observed upto 90 minutes of post ictal duration, but was more marked when the post ictal duration was < 30 minutes. In the Group I with post ictal duration of < 30 minutes, the mean serum prolactin level was 33.16 + 14.6Ing/ml and with post ictal duration of > 30 minutes, the mean value was 20.04 + 6.75 ng/ml. In the Group II, these values were 11.84 + 2.74 ng/ml and 11.14 + 2.52 ng/ml respectively, and in

Table IV: Serum prolactin level in three groups in relation to post ictal duration (<30 minutes vs.>30 minutes)

Variables		Group I (n=30)	Group II (n=20)	Group III (n=20)
Post ictal duration <30 min	Mean± S.D.	33.16 ± 14.61 (n=19)	11.84 ± 2.74 (n=12)	9.05 ± 3.17 (n=9)
Postictal duration >30 min	Mean± S.D	20.04 ± 6.75 (n=11)	11.14 ± 2.52 (n=8)	8.46 ± 2.89 (n=11)
	t-value	10.092	0.939	0.756
	d.f.	28	18	18
	p-value	<0.0001**	0.3	0.4

** Highly significant (p<0.0001)

the Group III, these values were 9.05 + 3.17 ng/ml and 8.46 + 2.89ng/ml respectively. Statistical analysis revealed that the values were highly significant (p<0.001) only in the group I and not significant in group II & III (p>0.3, >0.4 respectively; Table IV).

Discussion:

Epilepsy is the commonest neurological condition of the childhood. It is often confused with other frequently occurring non epileptic paroxysmal disorders of the childhood. Acute changes in the pituitary hormone levels, which occur following some of the seizures can help in differentiating epileptic seizure from pseudoseizure and febrile seizure. The most predictable post ictal changes are increased serum cortisol levels and serum prolactin levels. Because of the normal diurnal variation in serum cortisol levels and the relative delay in the post ictal elevations of serum cortisol, serum prolactin level is more useful as diagnostic measure of epileptic seizure. In the present study, significant elevation of serum prolactin level was observed only in children of Group I (28.77±15.49 ng/ml) as compared to control (9.53 ± 2.45 ng/ml). Serum prolactin levels were higher in GTCS (35.06 ± 14.26 ng/ml) and CPS (28.73 ±12.94 ng/ml) as compared to SPS (13.74 ± 6.92 ng/ml).

Similar observations were also made by many other workers showing increase in serum prolactin levels post ictally especially after GTCS. Abbott et al (1980) had demonstrated that elevation of serum prolactin level is not due to a non specific response to stress but probably indicates an alternation in the hypothalamic neurotransmitter activity during the seizure.

Collinas et al (1983), Rao et al (1989) and Graf et al (1988) had also reported similar results, but Lusic et al (1999) had found confounding results in patient with seizure and syncopal attacks.

An elevation of serum prolactin can be taken as a predictor of epilepsy. A non elevation of prolactin is not predictor of epilepsy and hence does not rule out the diagnosis of epileptic seizure as non elevated levels were seen in up to 20% of GTCS, 38% CPS and 72% of SPS.

Sperling & Wilson (1986) found that complex partial seizures were associated with bilateral limbic ictal discharges and had a significant rise in the serum prolactin concentration. Cases which did not exhibit a rise in serum prolactin levels, ictal discharges probably originate in the frontal and supplementary motor cortex without involving the limbic system. It has been suggested that when ictal discharges spread from the medial temporal area to the hypothalamic nuclei, they also lead to an alternation in consciousness. This probably explain why more cases of GTCS and CPS had elevated levels of prolactin. In SPS the decreased intensity and spatial involvement, probably accounts for the decreased incidence of prolactin elevation.

Culebras et al (1987) have studied the response of prolactin to seizures and to stress and reported that stressed patients had significantly less elevated prolactin levels, suggesting that neurogenic stimuli responsible for post ictal release of prolactin is independent of stress mechanism.

Wyllie et al (1984) reported marked prolactin elevation above three times of baseline at 15 or 30 minutes post ictally in 80% GTCS, 43% of CPS and 10% of SPS. Bauer et al (1989) found significant rise in serum prolactin level in 88% of GTCS, 78% of CPS and 22% of SPS. Observation of the present study are at par with the above studies. Zelnik et al (1991) observed significantly higher prolactin levels in the epileptic group (26.5 ±3.3 ng/ml) as compared with children with febrile seizures (13.2± 1.0 ng/ml), fever (11.2 ± 0.9 ng/ml), syncope (7.3 ± 0.9 ng/ml) and the control group (7.9 ± 0.6 ng/ ml). Pritchard et al (1985) reported a two fold increase in prolactin level following

true epileptic seizures, but no significant change occurred after pseudo epileptic attacks.

Kurlemann et al (1992) studied serum prolactin level in cerebral and psychogenic seizures in childhood and adolescence. A more than 2 to 3 fold prolactin increase of the baseline value occurred almost always after grandmal seizures, and regularly after complex partial seizures, but no hyperprolactenemia was observed after psychogenic seizures.

Malkowicz et al (1995) reported that seizure occurring after longer seizure free interval showed robust prolactin responses. After shorter seizure free interval prolactin response was reduced.

As Shown in table III, children with febrile seizure had higher prolactin levels than children with non epileptic events and controls, but the levels were still with in the normal range. Similar results were also observed by Zelnik et al (1991) and Dirik et al (1996).

In children with febrile seizures, minor rise in serum prolactin level was found, this is because of non specific response to fever related stress. Petroni et al (1998) had also reported similar results in febrile and true seizures.

In children with conditions mimicking seizures, no rise in the serum prolactin level was observed. Similar results were also observed by Graf et al (1988).

Maximum elevation of prolactin is seen within 15 to 30 minutes post ictally but levels can be assessed up to 90 minutes of post ictal duration. This is significant as it is not always possible to obtain a sample within the period of maximum elevation. As the prolactin levels are normal after two hours of post ictal duration, the test loses its significance and can not be used to differentiate true epileptic event from other events.

Conclusion:

An elevation of serum prolactin can be taken as a predictor of epilepsy. Maximum elevation of prolactin is seen with in 15 to 30 minutes post ictally but levels can be assessed up to 90 minutes of post ictal duration .It is suggestive that GTCS or CPS has occurred.

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Source of Support : Nil.
Conflict of Interest: None declared.

Case Report

Sternoclavicularis - A variant of Pectoralis Major Muscle

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Abstract:

A rare muscle sternoclavicularis was found in a large triangular gap between the sternocostal and clavicular heads of Pectoralis Major muscle on the right side during routine cadaveric dissection. Sternoclavicularis was seen to arise from the anterior surface of manubrium sterni and the capsule of sternoclavicular joint and was inserted on the anterior surface of middle one third of the clavicle. It was supplied by the lateral pectoral nerve. Sternoclavicularis muscle may help in stabilizing the clavicle and may partially fill the triangular deficit in the origin of the Pectoralis Major. This variation may be of particular interest to plastic surgeons, orthopaedic surgeons, radiologists and neurologists. It may mimic a tumour at this site.

Key Words: pectoralis major, sternoclavicularis muscle, anatomical variation

Introduction:

The pectoralis major muscle (PM) is a thick triangular muscle that usually arises from the medial half of the anterior surface of clavicle, the sternum, and the upper six costal cartilages and the upper part of the aponeurosis of external oblique muscle of abdomen. These three heads, the clavicular, sternocostal and abdominal, combine to form a tendon that inserts into the lateral lip of bicipital groove of humerus. The pectoralis major muscle is innervated by the medial and lateral pectoral nerves (Johnson & Ellis, 2005).

A number of variations of PM have been reported in literature, such as partial or complete absence of sternocostal portion, accessory head arising from serratus anterior muscle, absence of abdominal slip and decussation of fibers across the midline (Kida et al, 2000; Mosconi & Kamath, 2003; Loukas et al, 2006; Johnson & Ellis, 2005). Presence of additional musculature in the pectoral region have also been reported such as sternalis (O'Neill et al, 1998); Pectoralis quadrats (Bergman et al, 1988; Bonastre et al, 2002) and Chondroepitrochlearis muscle (Loukas et al, 2005). In the present case report, a rare anomaly of the PM is reported and is discussed in the light of available literature.

Case report:

During routine dissection, a large triangular gap was observed between the sternocostal and clavicular fibers of right Pectoralis Major muscle (Fig. I). Further cleaning and dissection showed an anomalous muscle occupying the base of this triangular gap (Fig. II). It originated from the anterior surface of the manubrium and capsule of the sternoclavicular joint. The muscle was directed upwards and laterally, passing deep to the clavicular fibers of the Pectoralis major. It was inserted on the anterior surface of middle one third of the clavicle, and was supplied by the lateral pectoral nerve. The subclavius muscle was normal and was placed deep to this anomalous muscle (Fig. III).

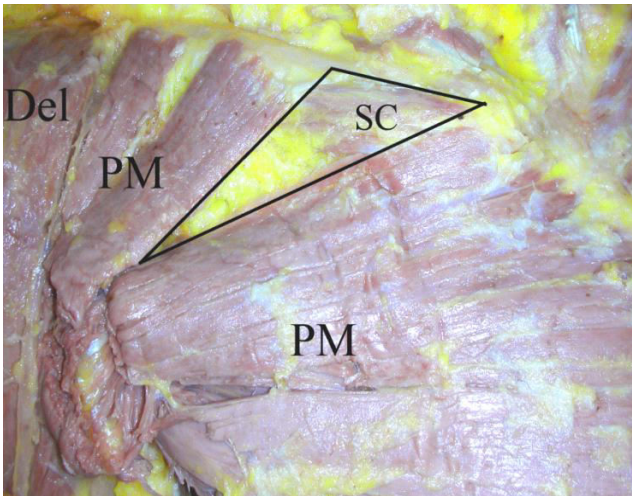

Fig. I: Right pectoral region showing triangular deficit between sternocostal and clavicular head of Pectoralis Major muscle.

Discussion:

Number of variations of PM and presence of supernumerary muscle in the pectoral region have clinical importance. The existence of these variants

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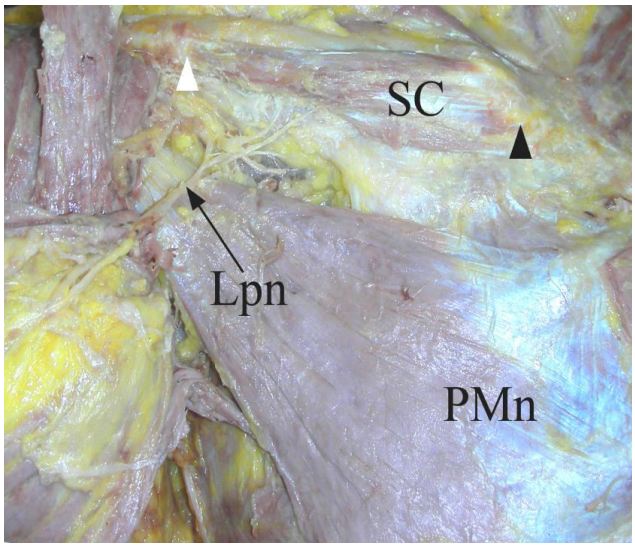

Fig II: Sternoclavicularis muscle originating from the anterior surface of manubrium streni and capsule of sternoclavicular joint (Black arrow head) and inserted on the anterior surface of middle part of clavicle (White arrow head). A branch of lateral pectoral nerve (Lpn) is seen entering this muscle.

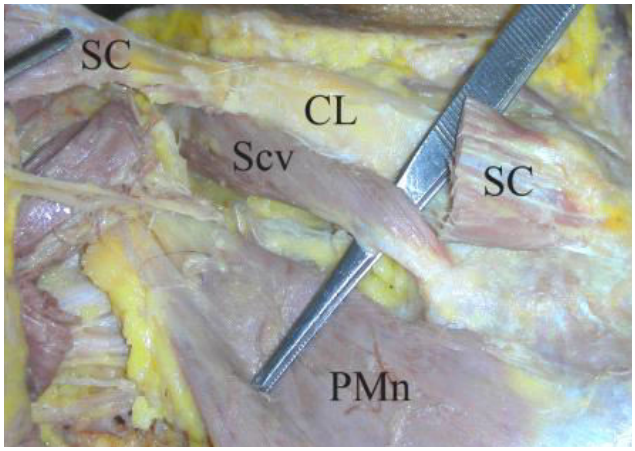

Fig. III: Subclavius muscle (Scv) was normal in its disposition lying deep to sternoclavicularis muscle (SC) which has been cut and retracted. *(Abbrevations used in Figur I, II & III: PM - Pectoralis Major; Del-Deltoid; SC - Sternoclavicularis; PMn- Pectoralis minor; CL - Clavicle)*

can be explained by reviewing its development. Molecular studies indicate a crucial role of the fibroblast growth factors (FGFs) in the limb initiation and role of *Hox*genes in the differentiation of somites and regulation of cell proliferation (Larson, 2001). Fibroblast growth factors from the apical ectodermal ridge of developing limb activates zone of proliferating activity, which causes expression of the sonic hedgehog genes. Molecular studies show that sonic hedgehog genes secretions control the patterning of the limb (Moore & Persaud, 2003). The pectoral muscles assume their final forms through a combination of migration, fusion and apoptosis of myoblasts of pectoral sheet (Carlson, 2004). The pectoral sheet divides into superficial ectopectoral layer and deep endopectoral layer (Schafer et al, 1923). The subclavius is regarded as a derivative of the deep layer of the pectoral sheet. The outer lamina is divided into clavicular, presternal and abdominal portions. The sternoclavicularis (SC) arises from the border of presternum and is inserted into the anterior surface of clavicle, as far as or even beyond, the middle third of the bone (Schafer et al., 1923). Supernumerary muscle in the present case report represents the SC. Presence of SC and triangular deficit of PM may be a result of the failure of designated myoblasts of ectopectoral fascia to undergo proper orientation and their subsequent degeneration.

The SC is a rare muscle and has not been described amongst the Indian population. A clinical problem could arise if sternoclavicularis was to be mistaken for a mass or tumour during CT or MRI. The presence of the SC may have positive functional implications. The SC may help in stabilizing the clavicle during various movements. It may effectively pull forward the lateral part of the clavicle which may enhance the functional activity of clavicular part of the PM and may compensate for the triangular deficit of the pectoralis major. This case report illustrates the need for continued reporting of anatomical variations and also their functional and clinical significance.

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Case Report

Desmoplastic Fibroma of the Maxilla : Report of a Rare Case

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Abstract:

Desmoplastic fibroma (DF) is a rare, benign but locally aggressive, intra-osseous lesion with a high tendency of local recurrence. A rare case of desmoplastic fibroma in the maxillary corpus is being presented with its pathology, clinical diagnostic methods, treatment and prognosis.

Key Words: Desmoplastic, Fibroma, Maxilla.

Introduction:

Desmoplastic fibroma is a benign, locally aggressive, intra-osseous tumor that rarely involves the facial bones. It represents the intra-osseous counterpart of the soft tissue fibromatosis or desmoid tumor. Jaffe & Selin in 1951, were the first to use the term desmoplastic fibroma to describe the lesion as a separate fibrous tumor of the bone. The first report about a desmoplastic fibroma of the jaw was presented by Griffith & Irby in 1965. In jaw area, non-odontogenic fibromatosis was declared as desmoplastic fibroma which distinguished it from Odontogenic fibroma (Slootweg & Muller, 1983; Depprich et al, 2005).

It is an extremely rare tumor with less than 200 cases in the published literature with a reported incidence of 0.11% to 0.13% among primary bone tumors (Bohm et al, 1996). It occurs more often in the first 3 decades of life with equal occurrence in men and women (Crim et al, 1989).

As therapy, surgical resection, radiotherapy and if necessary, pharmacological treatment are recommended. Due to high recurrence rate, surgical resection is the most preferred option (Sinno & Zadeh, 2009; Ikeshima & Utsunomiya, 2005).

In this report we present the clinical course and treatment of a patient with the diagnosis of desmoplastic fibroma in the right maxillary corpus, which itself make it rarest of the rare occurrence.

Case Report:

A 17-year-old male patient reported to the Department of oral and Maxillofacial Surgery with the complaint of swelling over his right cheek causing facial asymmetry. Patient gave the history of swelling being a pea sized growth since childhood which gradually increased in size with age. The swelling was not associated with any pain or discharge, and the patient was mainly concerned with the distortion of the face caused by the swelling. On inspection, a solitary oval swelling with diffused margins of approximately 5 X 6 cm was seen extending from the right angle of mouth to 1cm lateral to right angle of mandible mesio-distally, and extending from 1cm below the right zygomatic butress till lower border of mandible. On palpation, the swelling was found to be firm in consistency, lobulated, non-tender and fixed to underlying structure with no rise in local temperature.

Intra oral examination revealed an exophytic lobulated oval mass measuring 3 X 2.5 cm and hanging from buccal alveolar mucosal aspect in relation to 15, 16 & 17 (Fig. I). Vestibular obliteration was present with respect to right buccal mucosa. On palpation, it

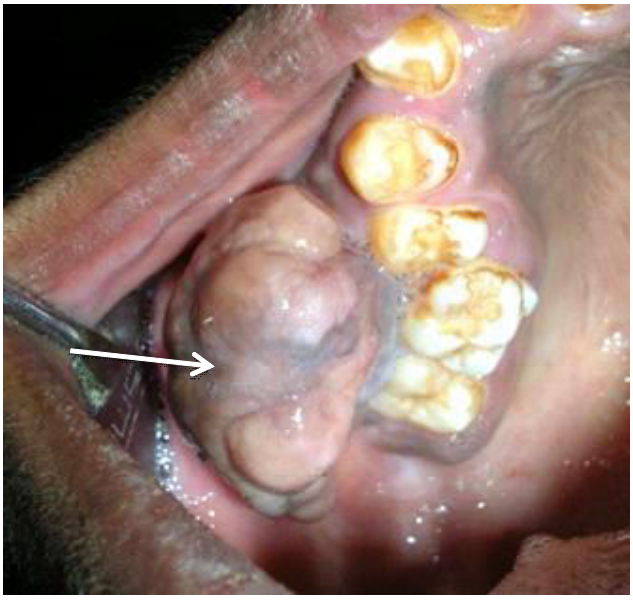

Fig.I: Exophytic lobulated mass seen over right buccal mucosa.

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was lobulated and firm in consistency. Superiolateral aspect in relation to buccal vestibular area of 16, 17 had hard bony mass fixed to underlying structure, displacing 15, 16, 17 palatally.

Orthopantomogram did not reveal any gross bony change. Computerized tomography scan revealed, a fibro osseous growth over right maxilla, just posteriolateral to molar teeth causing subcutaneous elevation of soft tissue and the lesion was centrally located (Fig. II). Three Dimensional CT reconstruction revealed expansion of right maxilla anteriorly.

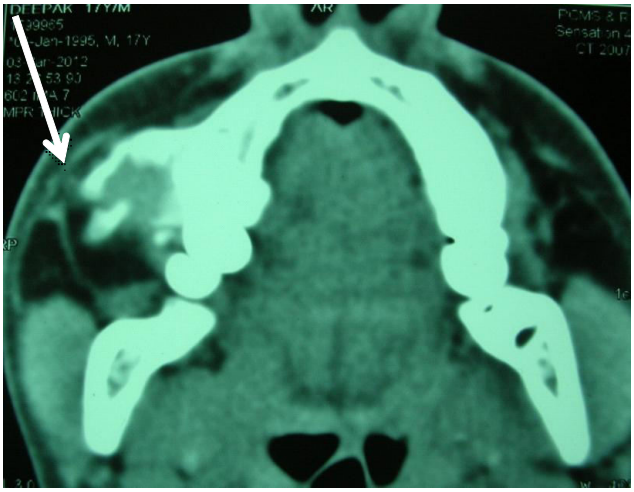

Fig.II: CT scan revealed fibro osseous growth over right maxilla.

Fine needle aspiration cytology was negative and therefore, incisional biopsy was done which was suggestive of desmoplastic fibroma. Routine blood and biochemical examination did not reveal any significant changes. Surgical resection of the tumor and curettage of the peripheral bone was planned under GA.

The tumour was excised and 17,18 were extracted because of its direct association with the tumour mass. Maxillary bony expansion was reduced using vulcanite burr and bone files and primary closure was achieved.

This tumour showed a very slight adherence to the surrounding bone and was resected easily. The histopathological examination of the resected mass revealed spindle-shaped cells with myofibroblastic differentiation, abundant collagen formation and low proliferation activity (Fig. III).

Based on these characteristics, the diagnosis of a desmoplastic fibroma was made. The post-operative recovery was normal. One year follow up of the patient showed no recurrence (Fig. IV).

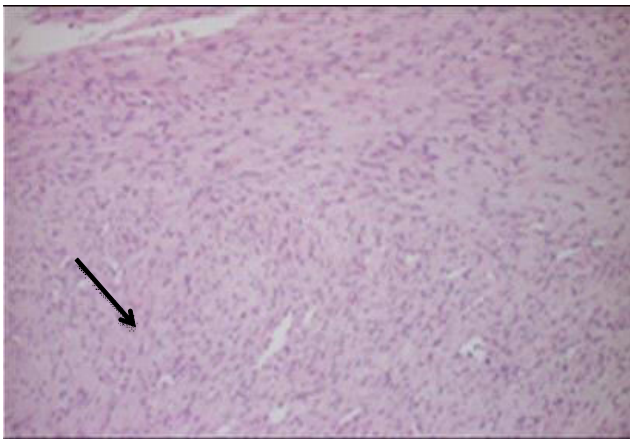

Fig.III: Partially parallel or plexiform arranged spindle cells with slim and elongated nucleus without cytological signs of malignancy. High- grade formation of collagen fibre. (H & E Stain, 100X).

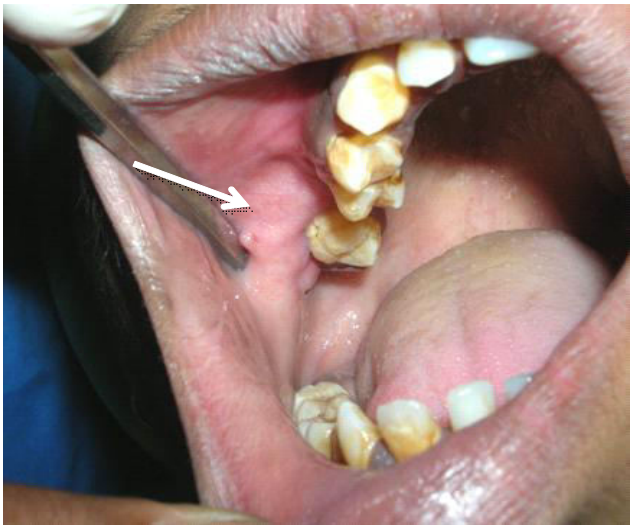

Fig.IV: Patient after 1 year follow up.

Discussion:

Desmoplastic fibroma of the maxillofacial region may be difficult to diagnose early in the disease process because of a slow, insidious onset coupled with unremarkable radiographic findings. It is a disease of young people, with a peak incidence in the second decade of life (Templeton et al, 1997).

Rabhan & Rosai (1968) and Kwon et al (1989) reported that a desmoplastic fibromas of the jaw with increased cellularity have a higher tendency to recur. Controversy exists regarding the preferred method of surgical management. Jaffe & Selin (1951) recommended segmental resection, but also stated that thorough curettage was an acceptable alternative.

Freedman et al (1978) found that 19 of 22 cases of desmoplastic fibroma had no evidence of recurrence, with follow-up time ranging from 3 months

to 8 years. He concluded that curettage was the preferred treatment.

In the present case, excision of the tumour mass with curettage of surrounding bone was the preferred treatment because of non-aggressive nature of the lesion demonstrated by slow progression of the swelling over a duration of 17 years and the elevation of soft tissue rather than extension radiographically. Isolated intra-osseous lesion without evidence of extension into contiguous soft tissue or perforation of cortical bone may be adequately managed by thorough curettage. However, when a lesion displays signs of aggressive behaviour and extension into soft tissue, segmental resection should be considered.

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Case Report

Concomitant Pseudoaneurysm and Arteriovenous Fistula Formation of Brachial Artery: A Case Report

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Abstract:

Concomitant pseudoaneurysm and arteriovenous (AV) fistula formation of brachial artery after a penetrating injury has been rarely reported. False aneurysms of peripheral arteries are very rare. It is usually of infectious, post-traumatic or of iatrogenic etiology. In most cases, these are the result of penetrating injuries such as gunshot or stab wounds and iatrogenic arterial injuries. Brachial artery is not a common site for peripheral artery aneurysms. Its association with fistula formation with adjacent vein makes this a rare case. This case was diagnosed on colour duplex ultrasound and confirmed on subtraction angiography.

Key Words: Pseudoaneurysm, Brachial artery, Arteriovenous fistula.

Introduction:

Post-traumatic pseudoaneurysm development is very rare in the peripheral arteries and is generally a late sequelae of trauma. The frequency of peripheral artery pseudoaneurysms is less common in the upper extremity as compared to the lower extremity (Wielenberg et al, 2000). Popliteal artery is comparatively a common site in lower extremity, but brachial artery aneurysm is an uncommon entity. Only few cases of brachial artery pseudoaneurysms have been reported in the medical literature to date but no case has been reported of its association with AV fistula. Their diagnosis and surgical treatment are extremely important, because they can cause severe disability, including loss of upper extremity and hand.

Here we report a case of a young man who came to us for colour duplex sonography and digital subtraction angiography for swelling in distal arm developed after trauma. We diagnosed it as post-traumatic brachial artery pseudoaneurysm with arteriovenous fistula formation, along with retained foreign body.

Case Report:

A 32 year old male was referred to us from the department of Surgery for colour duplex sonography and digital angiography, with history of a progressive painless swelling on the medial side of left distal arm for 3 month. He gave a history of penetrating trauma by an iron rod while cutting it, which caused a profusely bleeding wound. He took primary treatment in his local area. Later on he developed a painless gradually increasing swelling which did not cause any impairment in the functional capacity of the limb (Tetik et al, 2002). Only his apprehension about the nature of the swelling brought him to the hospital. He did not gave any history of fever, numbness or tingling sensation at the site of swelling or palpitation. There was no history of venepuncture, arteriography, dialysis, intravenous drug abuse or surgery at the site of swelling. There was no personal or family history of diabetes, hypertension, connective tissue disorder or history of aneurysm.

On physical examination, a widely pulsatile ovoid mass was present on the medial side of the left distal arm, which was approximately 5 cm in size, with an old scar mark at the site of the swelling. There was no visible pulsation, skin pigmentation or prominent veins. Palpation of the swelling revealed a soft, compressible, mobile, pulsatile, expansile and non fluctuant mass. It was non tender, non reducible, not blanching on pressure and not attached to overlying skin or underlying muscle or bone. Cardiovascular and neurological examination was unremarkable. There was no evidence of embolisation to the digits; pulses were palpable distal to the swelling. The remaining physical examination was normal. The laboratory examination was normal. Plain X-ray showed a metallic foreign body in left distal arm.

On ultrasound, two anechoic collections of approximately 3.4×2.0 cms and 5.4×2.4 cms size were seen along the distal part of brachial artery which

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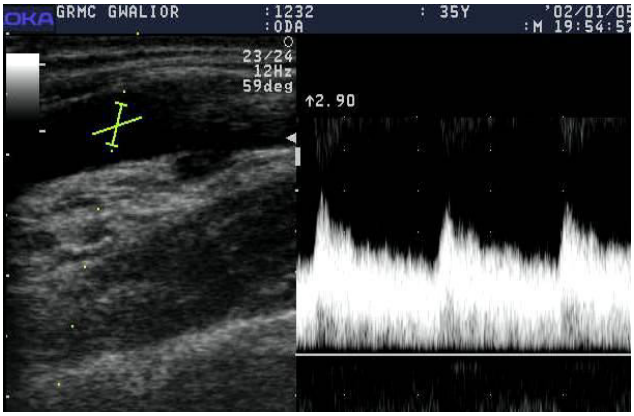

Fig. I: Spectral tracing shows high diastolic flow in brachial artery in proximal part which is an indirect evidence of fistula located downstream.

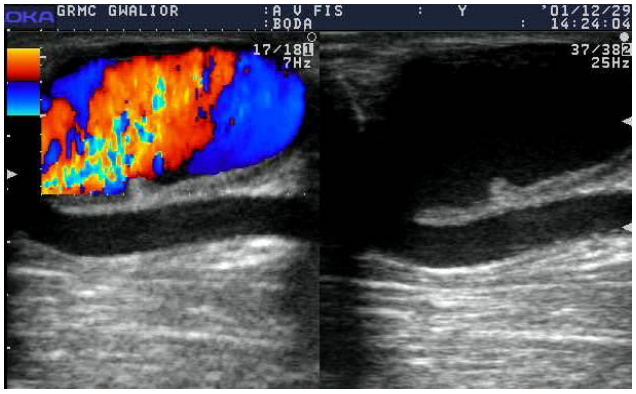

Fig. II: Colour duplex sonography of brachial artery pseudoaneurysm: shows yingyang pattern caused by swirling of blood in the pseudoaneurysm cavity.

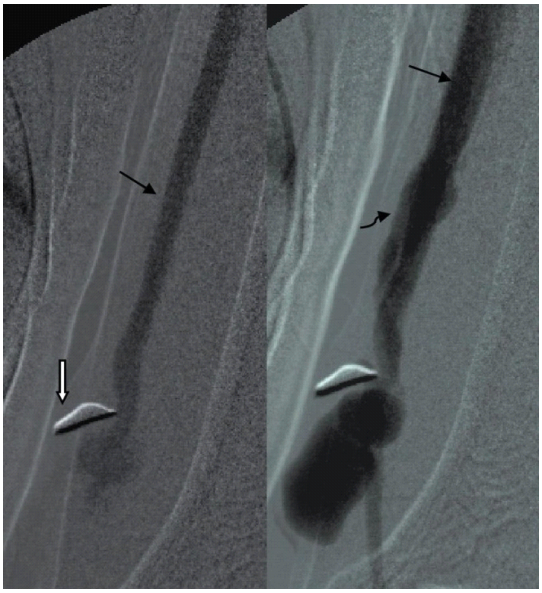

Fig. III: Selective digital subtraction angiography of brachial artery:(A) early arterial phase: filling of brachial artery (black arrow) with a small pool of contrast along the artery;(B) late arterial phase: showing a dilated, tortuous vein(curved arrow) alongside the brachial artery which is filling soon after injection of contrast. Two pools of contrast seen along the brachial artery. A metallic foreign body(white arrow) was seen adjacent to the pseudoaneurym.

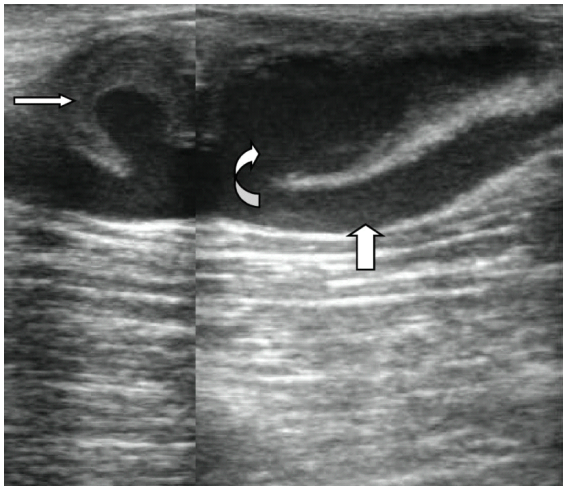

Fig. IV: Gray scale sonography of brachial artery: longitudinal image of brachial artery (thick arrow) shows two perivascular fluid collections(thin and curved arrow). Note the defect through which artery and collections are communicating.

were communicating directly with the artery. On colour doppler, these lesions were showing 'yin yang' pattern caused by swirling of blood in these cavities. The spectral tracing showed classic to-and-fro waveform pattern (turbulent flow). Along the side of these perivascular collections, a dilated anechoic vessel was seen which was also communicating directly (fistulous communication) with the brachial artery which was confirmed on high colour velocity settings (high colour pulse repetition frequency). This was a vein showing arterialised signals on doppler. The brachial artery above the fistula was showing relativly high diastolic flow on doppler which is an indirect evidence of a fistula located downstream. Evaluation of the abdominal aorta, femoral and popliteal arteries with duplex ultrasound identified no other aneurysm.

Selective digital subtraction angiography showed a dilated, tortuous vein along the side of brachial artery which was filling soon after injection of contrast in the brachial artery. The vein filled up immediately after contrast reached the distal part of the brachial artery and before it could reach the radial or ulnar artery, which indicates a communication between the brachial artery and the vein. Two separate pools of contrast were seen along the brachial artery, suggesting aneurysmal dilatation from the artery. A metallic foreign body was seen in the distal arm adjacent to the pseudoaneuryms.

Discussion:

Arteriovenous fistula is an abnormal connection

or passageway between an artery and a vein. It may be congenital, surgically created for hemodialysis treatment, or acquired due to pathologic process, such as trauma or erosion of an arterial aneurysm (Fatimi et al, 2010.

Aneurysms at less common locations are generally due to major trauma, syphilis, Marfan syndrome or infection. Aneurysms can develop in all arteries of the human body. Atherosclerotic aneurysms are often seen in large arteries and in patients of advanced age, but pseudoaneurysms due to penetrating or blunt trauma are seen in patients of every age and at any location (Ho et al, 1987). Frequency of pseudoaneurysms in the upper extremities is much lower than that in lower extremities.

A pseudoaneurysm, also known as a false aneurysm, is a hematoma that forms as the result of a leaking hole in an artery. Note that the hematoma forms outside the arterial wall, so it is contained by the surrounding tissues. Also it must continue to communicate with the artery to be considered a pseudoaneurysm. This must be distinguished from a true aneurysm which is a localised dilatation of an artery including all the layers of the arterial wall. A pseudoaneurysm is also different from an arterial dissection, which is a separation of the layers of the arterial wall, and may be associated later with aneurysm formation. Distinctively, in a pseudoaneurysm, the hole in the arterial wall is generally the consequence of a iatrogenic trauma, most likely a previous invasive medical procedure that necessitated intrusion into an artery, for example to place a stent. Alternatively, a pseudoaneurysm can also occur as a complication of acute pancreatitis, due to enzymes leaking out from the pancreas and damaging nearby vessels. In contrast true aneurysms and dissections are usually the consequence of congenital or acquired deficiency in the arterial wall, for example due to atherosclerosis.

Infection, polyarteritis nodosa, congenital arterial defects, and especially trauma play a role in the pathogenesis of upper extremity pseudoaneurysms. Atherosclerotic aneurysm of the brachial artery is very rare (Napolitano et al, 1998). If the only causal factor is trauma, the aneurysm takes the form of a pseudoaneurysm. Minor blunt trauma may cause pseudoaneurysms in patients who are prone to haemorrhage. If no neurologic or thromboembolic complication develop, aneurysms of 2 cm or less in diameter can be silent or asymptomatic for a long period. Such aneurysms can be diagnosed easily by detailed medical history and physical examination. Sometimes patients are admitted to hospitals with pseudoaneurysms months or years after the trauma. Due to their clinical appearance, peripheral artery aneurysms of the extremities can be easily misdiagnosed as hematomas or even as soft tissue tumors. In addition, pressure and hyperemia can result in resorption of adjacent bone. A biopsy in such cases may be hazardous. The history of trauma (recent or even previous) in conjunction with progressive soft tissue swelling should alert the clinician to a potential vascular injury as a differential diagnosis. However, as life span is increasing and diagnostic and evaluation processes are improving, the detection of such pseudoaneurysms is becoming more common (Yilmaz et al, 1997). Differential diagnosis includes hematomas, pulsating tumours, AV malformation, lymphadenopathy, lipomas and abscesses.

Plain radiographs can show fracture if it is there and any displacement of the fracture fragment which can cause vascular injury. Colour-flow Doppler ultrasonography is a non-invasive, low-cost and easily available imaging method that can provide sufficient diagnostic information to plan the surgical procedure. Magnetic resonance imaging (MRI) can also be used; an aneurysm appears on both T1- and T2-weighted images and the use of intravenous gadolinium does not enhance the signal. Upper extremity arterial Doppler ultrasonography and magnetic resonance angiography can be used as diagnostic tools, but the gold standard is selective upper extremity arteriography (Johnston et al, 1991). Selective catheterization of the injured artery allows not only the detection of the aneurysm, but also the preoperative embolization if there is a feeding artery. Doppler ultrasonographic evaluation is sufficient for late postoperative follow-up evaluation.

Treatment for pseudoaneurysm that can be performed under colour-Doppler ultrasonographic guidance are, manual compression, ligation, endovascular graft implantation, embolization, ultrasound-guided thrombin injection, and surgical reconstruction.

Conclusion:

In conclusion, pseudoaneurysm distal to the axillary artery is rare and is frequently the result of a gunshot or stab wound. Concomitant brachial artery pseudoaneurysm and arteriovenous fistula formation

after a penetrating injury is rare (Fatimi et al, 2010. Axillary and distal peripheral artery pseudo aneurysms of the upper extremity are less dangerous than are thoracic and abdominal aortic aneurysms. However arterial doppler ultrasonography, magnetic resonance angiography and selective upper extremity arteriography (DSA- gold standard) are the modalities which are helpful in correct and timely diagnosis of the aneurysm. Treatment includes resection of the false aneurysm with end-to-end direct anastomosis.

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Case Report

Study of Placenta in HELLP Syndrome Patient: A Case Report

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Abstract:

HELLP syndrome is a multi systemic disorder that complicates pregnancy and has a poor prognosis. HELLP syndrome is frequently associated with severe pre-eclampsia or eclampsia but can also be diagnosed in the absence of these disorders .We report a case of 20 year old married pregnant female who was diagnosed as a patient of HELLP syndrome.

Key Words: HELLP syndrome, pre-eclampsia, placenta, Tenny parker change.

Introduction:

The acronym 'HELLP' was first coined by Weinstein in 1982. He described it as a syndrome consisting of hemolysis, elevated liver enzymes & low platelet count. It is a multisystemic disorder that complicates pregnancy and has poor prognosis. Its incidence is reported to be 0.5-0.9 % of all pregnancies, and in 10-20% of women with severe pre- eclampsia (Haram et al, 2009). HELLP usually occurs in Caucasian women over the age of 25 years (Padden, 1999). Some experts consider it as a variant of pre eclampsia but others believe that pre-eclampsia & HELLP syndrome are separate disorder with overlapping features.

Case Report:

A 20 years old married women presented to the Department of Obstetrics and Gynecology, Subharti Medical College, Meerut with history of amenorrhea for 9 months and pain in the right upper abdominal quadrant. Her obstetrical history was G_1 with 40 week pregnancy. Her blood pressure at the time of admission was 140/90 mm/Hg. Her laboratory reports revealed elevated liver enzymes and low platelets count.
Total Serium Billirubin - 9.2 mg/dl; SGOT (AST)-800IU/L; SGPT (ALT)-424; Alkaline Phosphate-336 and Platelet Count-30,000/mm3 .She delivered a live newborn weighing 3 kg and Apgar score of 7/10 at 1 minute.

After delivery the placenta of the patient was taken for gross and histopathological examination and fixed for 24 hours in 10 % formaline solution. On examination, the shape of the placenta was discoid with thickness of 2.56 cm in the centre. Weight of the placenta after trimming the membranes and cord was about 700 gm, number of maternal cotyledons were 16. Meconium covered major part of placenta; Hematomas were also seen over it. Umbilical cord was eccentric in position and was inserted about 4 cm from the margin.

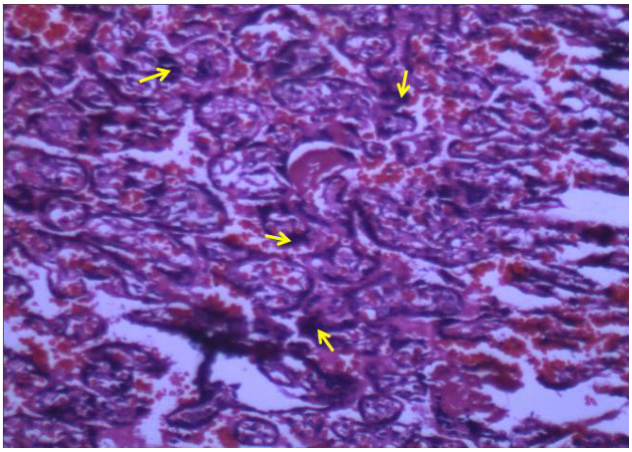

Fig. I: Photomicrograph of placenta showing matured chorionic villi and tenny parker change (H&E, 100X).

All the macroscopically detected focal changes in the placenta were sampled; histopathological examination revealed: Matured Chorionic Villi, Tenny parker change (Fig.I), Decidual arteriopathy, Thickening of vessel wall with fibrin deposits (Fig. II), Occlusion of vessel lumen by cellular thrombi & recanalization (Fig. III).

Discussion:

Generally the HELLP syndrome is considered a placenta instigated, liver targeted acute inflammatory condition with elements of disordered immunological processes. Like severe pre-eclampsia, it results from

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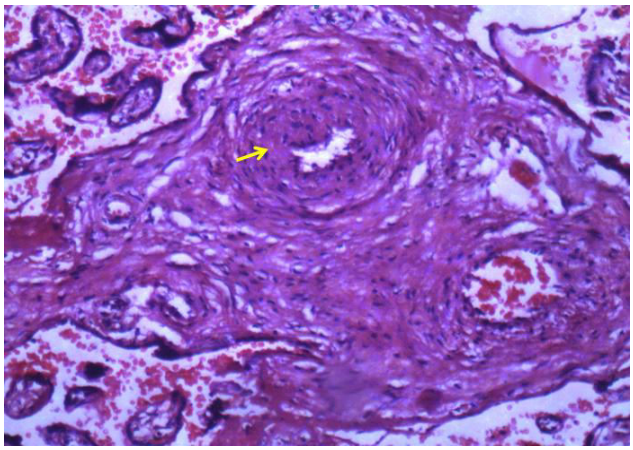

Fig. II: Photomicrograph of placenta showing thickened vessel walls with fibrin deposites (H&E, 400X).

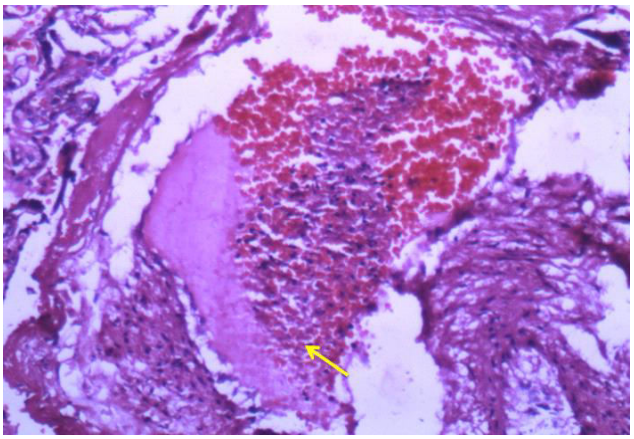

Fig.III: Photomicrograph of placenta showing thrombus and recanalisation in lumen of vessel (H&E, 100X).

the aberrant development, function and ischaemia of the placenta. Ischaemia of the placenta in turn triggers the release of factors that injure the endothelium via the loss of normal pregnancy vascular relaxation, release of vasoconstrictors and activation of platelets. Thus begins a cascade that is terminated only after delivery (Satpathy et al, 2009).

Histopathologic changes of the placenta in the present case, were in accordance with the results of study conducted by Vinnars et al (2008) to evaluate the histopathology in placenta from patients with severe pre-eclampsia with and without HELLP syndrome. There was significantly higher mean placental weight in HELLP syndrome group. Their histopathology showed evidence of accelerated villous maturation and decidual arteriopathy (Vinnars et al, 2008).

Tenny parker change is described as a presence of small size villi with increased syncytial knots. Syncytial knotting occurs as a part of normal villous maturation, but syncytial knots on more than 30% villi are indicative of perfusional compromise (Gersell & Kraus, 2002).

Decidual arteriopathy is defined as fibrinoid necroses of artery wall, often with dilation of the vessels, with or without the presence of acute atherosis or lumen thrombosis.

Smulian et al (2004) while examining placental lesions and birth weight in severe Pre-eclampsia and HELLP patients found no difference in weight and histopathology.

Raval et al (1997) investigating the maternal and neonatal outcome in severe PE and HELLP, did not found difference in birth weight.

Gul et al (2005) reported no differences in the percentage of intrauterine growth restriction between Pre-eclampsia and HELLP but reported a higher incidence of foetal mortality in HELLP patient.

Conclusion:

The prognosis of pregnancies complicated by HELLP syndrome depends on early diagnosis and early therapeutic approach. Patients with HELLP syndrome have a higher incidence of pre-eclampsia (43%) in subsequent pregnancies (Isler et al, 2003). After delivery, if the placenta is examined minutely it provides much insight into the prenatal health of the baby and mother. Special precautions can be instituted during antenatal period and labour in subsequent pregnancies to reduce further risk to the mother and foetus.

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Case Report

Collecting Duct Carcinoma of the Kidney

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Abstract:

Collecting duct carcinoma (CDC) is a rare, highly aggressive malignant neoplasm that arises from the collecting duct epithelium of the kidney. It generally pursues a more aggressive course than conventional renal cell carcinoma. The average age is approximately 53 years. These are large tumors commonly located in medulla or central part of kidney with extension into perinephric fat and invasion into renal pelvis. Microscopically, they show combined tubulo-papillary, microcystic and solid growth pattern; cells are highly atypical with a basophilic or eosinophilic cytoplasm and polymorphic nuclei, often of the hobnail type. Stromal desmoplasia and dysplastic changes in the neighbouring medullary renal tubules are often associated. Their biologic behaviour is mostly aggressive with a high rate of local, lymphatic and haematogenous spread at the times of diagnosis and a poor long-term prognosis.

Key Words: Collecting duct; renal cell carcinoma.

Introduction:

Collecting duct carcinoma (CDC), also known as Bellini duct carcinoma, is a rare tumour and constitute less than 1% of renal epithelial tumors (Matz et al, 1997). Although earlier cases had been reported but it was not recognized as a separate clinicopathological entity until 1986, when Fleming & Lewi (1986) described the clinical and morphologic features of six cases. Collecting duct carcinoma may occur at any age, the mean age at presentation is 53 years with a range of 13 to 83 years. Hematuria is the most common symptom followed by pain, weight loss and the presence of a palpable mass.

Case Report:

A 75 years old male presented with bleeding per urethra since 15 days and dribbling of urine since 8 days. On examination, in right renal angle a tender, firm to hard mass was palpable which measured around 12x6x5cms.

Routine investigations: Blood: Hb - 8.1gm/dl, TLC: 12,800, neutrophils-92%, lymphocytes-8%, ESR-135mm/hr; Urine: Reddish in appearance and turbid; pus cells-18-20/hpf, RBCs-plenty/hpf; Blood urea-88.2mg/dl and Serum creatinine-2.7mg/dl.

Special investigations: X-ray (KUB region) revealed a radio-opaque density in right renal region. Intravenous pyelography showed renal mass with calculi in right lower calyx. Ultrasonography real time scanning showed enlarged right kidney with hydronephrosis and isoechoic mass in lower pole calyx with calculus. Computerized Tomography scan of abdomen: hyperechoic mass within collecting system in right kidney with calculus in lower pole calyx and hydroureter.

On Gross examination of right nephrectomy specimen, it was found to be of greyish white in colour with irregular surface and variable sized nodules. It measured 12x6x5cms with attached fibrofatty tissue. Renal pelvis was not identified externally & ureter was also not seen. Cut section showed a large irregular greyish white solid tumour one side of the kidney measuring 8x6cms; renal cortical thickness measured 1cm; tumor was completely replacing hilum of the kidney; renal vessels and ureter could not be identified. One dark brown renal stone was seen within pelvis measuring 1cm in diameter and was adherent to kidney. No lymph node was identified in perirenal fat (Fig. I). Histopathological Examination: It showed a poorly circumscribed mass composed of highly pleomorphic epithelial cells arranged in ducts, tubules and at places with papillary pattern (Fig. II A & B). These cells were round to oval with abundant eosinophillic cytoplasm, vesicular nuclei with irregular nuclear membrane and prominent 1-2 nucleoli (Fig. III). Occasional mitosis seen. At places hobnail pattern was seen. Adjacent renal parenchyma revealed features of chronic pyelonephritis. A diagnosis of Collecting duct carcinoma with chronic pyelonephritis, hydronephrosis & nephrolithiasis of right kidney was made.

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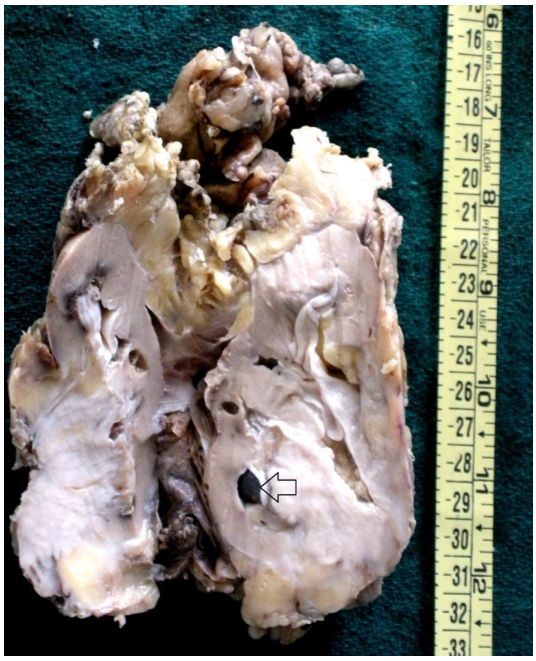

Fig. I: Cut section of mass shows large irregular grey white solid mass which is completely replacing the hilum.

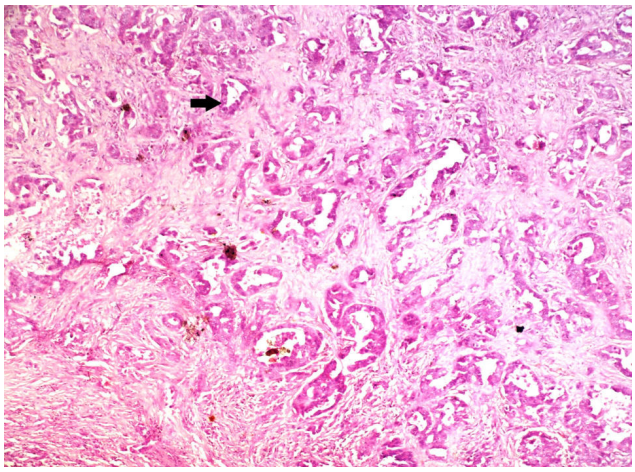

Fig. IIA: Section shows poorly circumscribed mass composed of pleomorphic cells arranged mainly in ducts and tubules (40x, H&E).

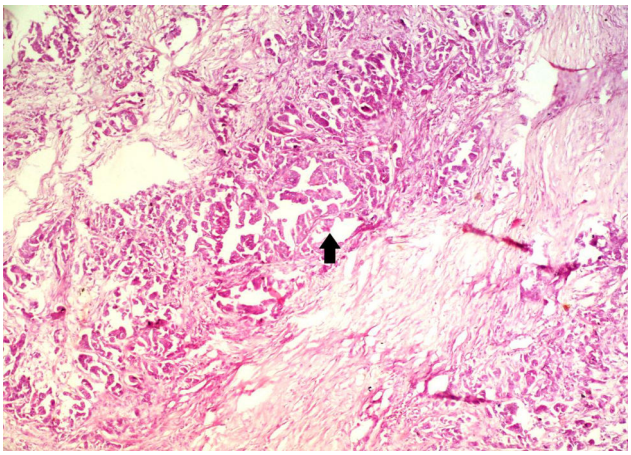

Fig. IIB: Section shows poorly circumscribed mass composed of pleomorphic cells at places in papillary pattern (40x, H&E).

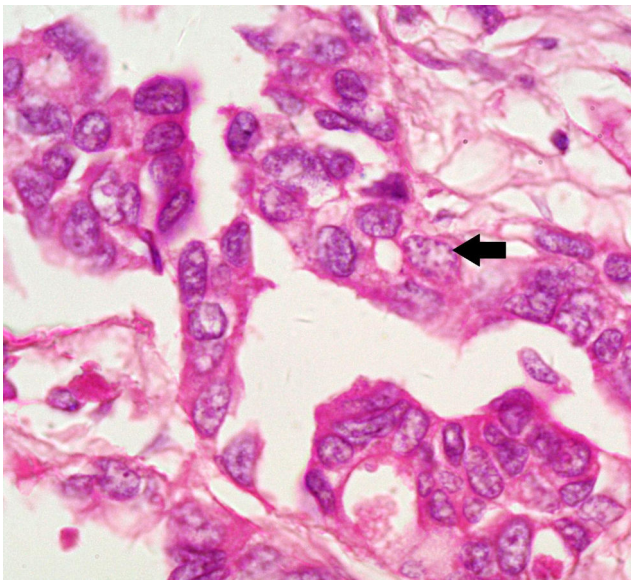

Fig. III: These cells are round to oval with abundant eosinophillic cytoplasm, vesicular nuclei with irregular nuclear membrane and prominent 1-2 nucleoli. (100x, H&E).

Discussion:

Collecting duct carcinoma (CDC) is a rare but distinct subtype of renal cell carcinoma; about 100 cases have been described in the literature so far (Srigely & Eble, 1998). Association with nephrolithiasis is extremely rare (Qureshi, 2007).

The first description of collecting duct carcinoma was given by Mancilla- Jimenez et al (1976) who reported a series of 34 papillary renal cell carcinomas (Kru lin et al, 2001). They observed atypical and hyperplastic changes in adjacent collecting tubules in three tumors, and hypothesized on their collecting duct origin. In 1979, Cromie et al (1979) described a renal tumor composed of papillary, transitional and tubular cell component, and suggested its origin for collecting duct. Many case reports appeared in the literature later.

Collecting duct carcinoma arises from the collecting duct epithelium of the kidney and shares a common embryonic origin with renal pelvis and minor and major calyces (mesonephros) rather than with proximal nephron (metanephros).

Grossly, the tumour may commence at the cortico-medullary junction but due to its aggressiveness, can spread to the entire kidney and beyond at diagnosis. Unlike conventional renal cell carcinoma, CDC is not usually circumscribed, and only small to punctuate hemorrhagic areas are present.

Histologically, it is characterized by tubulopapillary pattern of growth, marked desmoplasia, inflammatory infiltrate, high-grade cytological features

with hobnail nuclei. These tumors often have a mixed papillary and infiltrative tubular architecture. The infiltrative component is associated with marked stromal desmoplasia. Foci of dysplasia, or carcinoma in situ, can be found in the adjacent collecting ducts in some cases. The tumors are of high nuclear grade, corresponding to Fuhrman grade 3 or 4. Some cases have been described with a urothelial carcinoma component.

On immunohistochemical studies, tumor cell positivity with antibodies to Ulex European agglutinin 1 lectin strongly suggests the diagnosis of CDC. The tumor is also positive to peanut agglutinin (PNA), vimentin, lysozyme, distal tubular marker EMA, and high molecular weight cytokeratin, and negative for proximal tubular markers (Matei et al, 2005).

The main differential diagnosis of collecting duct carcinoma includes papillary renal cell carcinoma, adenocarcinoma or urothelial carcinoma with glandular differentiation and metastatic carcinoma (Qureshi, 2007).

The prognosis is poor, more than 50% of the reported patients died within two years of presentation (Kru lin et al, 2001). The collecting duct carcinoma is characterized by being an aggressive entity with an unfortunate outcome in most patients (Çalli et al, 2004).

Conclusion:

Identification of the Bellini's duct carcinoma has important diagnostic and prognostic ramifications. There are no specific radiological findings of this entity. The diagnostic process should involve meticulous attention to the architectural, histologic and immunohistochemical findings.

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Review Article

Pulse Oximetry: A New Tool in Pulpal Vitality Testing

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(Received September, 2011) (Accepted December, 2012)

Abstract:

Over the years, the thermal and electrical tests have been considered to be a suitable means of assessing the vascularity and vitality of the tooth pulp. Pulse oximetry is an effective, objective, oxygen saturation monitoring technique broadly used in medicine for recording blood oxygen saturation levels. It can also be used in endodontics for differential diagnosis of vital pulps and necrotic ones. However, there are some limitations inherent in the technology of pulse oximetry, such as the effect of increased acidity and metabolic rate, which causes deoxygenating of hemoglobin and changes in blood oxygen saturation, also movements of the body or probe can complicate readings. This test produces no noxious stimuli, therefore, apprehensive or distressed patients may accept it more readily than routine methods. A review of the literature and a discussion of the potential application of this system in endodontics is presented.

Key Words: Pulp vascularity, pulse oxymetry, endodontics

Introduction:

Diagnosis is the art of identifying the problem and using scientific knowledge to determine the cause of the problem. The purpose of diagnosis in endodontics is to assess the condition of a tooth and to identify the cause of the discomfort. To determine the vitality of a pulp, the ideal tests used should be objective, painless, and reliable. Currently the most common tests for this purpose are sensibility tests (Kenneth et al, 2011). A major limitation of these tests is that they subjectively imply vitality through sensory responses.

An alternative method would be to evaluate the vascularity of the pulp. This would be a more precise test, given the essential role of the pulp circulation in maintaining the tissue health. The evaluation of circulatory status of the pulp has been proposed to assess pulp vitality. Oximetry, the measurement of oxygen bound to hemoglobin is an advance in anesthesiology. Pulse Oximetry is a well established, noninvasive, direct, completely objective method for measuring vascular health by evaluating oxygen saturation levels (Noblett et al, 1996).

Histroy:

The concept of pulse oximetry is not new. In 1935 Carl Matthes built the first device to continuously measure blood oxygen saturation *in vivo* by transilluminating tissue. He used two wavelengths of light, one of which was sensitive to changes in oxygen saturation and the other, which was in the infrared range, was used to compensate for changes in tissue thickness, hemoglobin content and light intensity. Although useful in following trends in saturation, the device had limitations as it was difficult to calibrate and absolute values could not be obtained.

Squire in 1940 devised a technique of calibration by compressing tissue to eliminate the blood. This was later incorporated in the first generation of pulse oximeters used in the operating theatres. In the early 1940s, Millikan coined the term "oximeter" to describe a lightweight earpiece to detect the oxygen saturation of hemoglobin, for use in aviation research to investigate high altitude hypoxic problems. Thus, the clinical utility of pulse oximeters was evident to researchers in the field more than half a century ago.

In 1964, a surgeon, Robert Shaw, built a self-calibrating ear oximeter, which was marketed by Hewlett Packard in 1970 for use in physiology and cardiac catheterization laboratories (Stephen et al, 1951).

The year 1972 marked the greatest step forward in monitoring oxygenation, ironically as an incidental finding. Until then, in order to isolate arterial blood for transillumination, oximeters relied on compression and heating the earlobe to remove signals from venous and capillary blood which often caused burns. Takuo Aayogi in 1974 at the Nihon Kohden Corporation working on a dye dilution cardiac output monitor using a ear densitometer, found artifacts due to pulsatile flow. He noted that the washout curves he was measuring, were modified by pulsatile variations. While attempting to eliminate these variations, he discovered that the absorbency ratios of these

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pulsations at different wavelengths varied with the oxygen saturation. Thus, he could minimize the pulsatile component by balancing the red light signal with an infrared light signal where the dye had no absorption. As this compensation was dependant on oxygen saturation, he incorporated the technique of reducing noise in his signal to measure oxygen saturation. The subsequent development of light emitting diodes (LEDs), photo detectors and microprocessors further refined the technique, and pulse oximeters were widely introduced into clinical practice.

Modern pulse oximetry was born with the realization that pulsatile changes in light transmission through living tissues are due to alteration of the arterial blood volume in the tissue. Measurement of the pulsatile component would eliminate the variable absorption of light by bone, tissue, skin, pigment, etc from analysis. The most important premise of pulse oximetry, therefore, is that the only pulsatile absorbance between the light source and the photo detector is that of arterial blood.

Two wavelengths of light are used; 660 nanometers (red) and 940 nanometres (near infrared). At 660nm, reduced hemoglobin absorbs about ten times as much light as oxyhemoglobin. At the infrared wavelength, (940nm), the absorption coefficient of oxyhemoglobin is greater than that of reduced hemoglobin. The pulse oximeter directly senses the absorption of red and infra red light, and the ratio of pulsatile to nonpulsatile light at the red and infrared wavelengths are translated through complex signal processing to a function of the arterial oxygen saturation. A microprocessor integrates the data, and through an elaborate calibration algorithm based on human volunteer data, the oxygen saturation can be estimated (Comroe & Botelho, 1947).

Principles of Oximetry:

In the 1930s Matthes used spectrophotometry to determine hemoglobin oxygen saturation. This method is based on the Beer-Lambert law, which relates the concentration of a solute to the intensity of light transmitted through a solution (Severinghaus & Astrup, 1987).

$I_{trans} = I_{in}e^{-A}$

$A = DC\epsilon$

Where I_{trans} =intensity of transmitted light; I_{in} =intensity of incident light; A = absorption; D= distance light is transmitted through the liquid (path length); C= concentration of solute (hemoglobin); ϵ = extinction coefficient of the solute (a constant for a given solute at a specified wavelength). Thus, if a known solute is in clear solution in a cuvette of known dimensions, the solute concentration can be calculated from measurements of the incident and transmitted light intensity at a known wavelength. The extinction coefficient is a property of light absorption for a specific substance at a specified wavelength. In a one-component system, the absorption A is the product of the path length, the concentration, and the extinction coefficient, equation la. If multiple solutes are present, A is the sum of similar expression for each solute. Laboratory oximeters use this principle to determine hemoglobin concentration by measuring the intensity of light transmitted through a cuvette filled with a hemoglobin solution produced from lysed red blood cells. For Beer's law to be valid, both the solvent and the cuvette must be transparent at the wavelength used, the light path length must be know exactly, and no absorbing species can be present in the solution other than the known solute. It is difficult to fulfill these requirements in clinical devices; therefore, each instrument theoretically based on Beers lambert law also requires empirical correction to improve accuracy (Jayashankar et al, 2012; Radhakrishna et al, 2002).

Equipment:

1. Pulse oximeter probe/ pulse oximeter sensor (POS):

It contains two light-emitting diodes (LEDs) - One transmits red light (approximately 660 nm) and the other transmits infrared light(900-940nm). It operates at 500 on/off cycles /sec.

2. Pulse oximeter monitor:

It gives digital display of oxygen saturation values,connects to POS.

3. Photo detector:

It detects the amount of light absorbed by oxygenated and deoxygenated hemoglobin and connected to a microprocessor.

There are no Oximetry probes specific for the teeth in the market.

Gopikrishna et al (2006) developed a custom made POS holder for an existing Nellcor multi-site sensor and showed the utility of the pulse oximetry dental probe in the assessment of human pulp vitality.

Mechanism of action:

The pulse oximetry sensor consists of two light emitting diodes, one to transmit red light (640nm) and the other to transmit infrared light (940nm), and a photo

etector on the opposite side of the vascular bed. The light emitting diode transmits light through a vascular bed such as finger, toes or ear. Oxygenated hemoglobin and deoxygenated hemoglobin absorbs different amount of red /infrared light. The pulsatile change in the blood volume causes periodic changes in the amount of red/ infrared light absorbed by the vascular bed before reaching the photo detector. The relationship between the pulsatile change in the absorption of red light and the pulsatile change in the absorption of infrared light is analyzed by the pulse oximetry to determine the saturation of arterial blood. The information collected is converted into digital signals that are processed by the oximetry computer. A numerical estimation of the hemoglobin oxygen saturation is then produced and displayed. The machines can produce audible and visible signals to alert the doctor to change in the pulse rate and oxygen saturation. The machines safety mechanism includes low oxygen saturation and pulse rate range alarms. Displacement of finger probe also causes an audible signal. The alarms can be set independently to desired range (Abd- Elmeguid & Yu, 2009).

The response to current clinical tests indicate only that the sensory fibers are vital and 10%-16% of the results of these test are false. The nervous system, which is highly resistant to inflammation , may remain reactive, even though all surrounding tissues have degenerated; therefore testing the sensory supply may give a positive response when the pulp is damaged i.e., false positive result (Petersson et al, 1999).

This test may also leave the patient with an unpleasant sensation. A false-negative result (i.e. no response) may be obtained in cases of calcific metamorphosis, recently traumatized teeth, and incomplete root formation (Mickel et al, 2006). The vitality of the pulp is determined according to the health of the vascular supply and not of the sensory fibers. The pulp receives its blood supply through thin-walled arterioles entering through the apical and accessory foramina. These arterioles run longitudinally through the centre of the pulp, branching out to its periphery where they form a capillary network in the sub-odontoblastic area. These capillaries do not enter the dentin; they drain into the venules that run alongside the arterioles and pass out through the same apical foramen. Different methods may be used to assess the blood flow in the pulp (Abd-Elmeguid & Yu, 2009). Advantages of Pulse Oximetry are that it is effective, objective, measures pulp vascularity, applicable to recently traumatized permanent teeth, non invasive, can be used in uncooperative, apprehensive patients, no unpleasant sensation, reproducible readings and data storage for further references (Radhakrishnan et al, 2002). There are some limitations inherent in the technology of pulse oximetry. They may be classified as intrinsic and extrinsic (Jafarzadeh & Rosenberg, 2009).

1. Intrinsic Factors: Increased acidity, increased carbon dioxide in the blood stream, increased metabolic rate arising from inflammation, intra venous dyes causing false low oxygen saturation level and presence of other gases such as carbon dioxide. For excellent accuracy, oxygen saturation level should be in the range of 70% to 100%.

a. Patient variables: Low peripheral perfusion, increased venous pulsation, hemoglobin disorders, pigmented patients vasoconstriction, hypotension, and body movement will cause false /delayed readings, extensively restored teeth, nail polish, if blue, green, or black causes inaccurate saturation readings

b. Environmental factors: Electro-cautery near the sensor, ambient light interferences, and ipsilateral blood pressure reading.

2. Extrinsic factors: Probe movement and overhead xenon arc lamps. The critical requirement for using pulse oximetry in dentistry is that the sensors should conform to the size, shape and anatomy of the tooth and that the LED and photo detector be parallel to each other and the probe should be held firmly .An innovative technological approach to reject motion artifact is termed as Masimo signal extraction technology (Kahan et al, 1996).

Laser Doppler Flowmetry (LDF) is another new innovation.It is an accurate, non invasive, reproducible, reliable method of assessing blood flow in microvascular systems with a diode that projects an infra red light beam through the crown and pulp chamber.Unfortunately Laser Doppler Flowmetry takes about an hour to produce recordings, making it impractical for dental practices (Polat et al, 2005).

On comparing Pulse oximetry *vs* conventional pulp sensitivity tests, Gopikrishnana et al (2006) found Pulse Oximetry to be more accurate.Sensitivity of the pulse oximetry was 100% ,cold test 81%, electric pulp test 71% in a study reported by Abd-Elmeguid & Yu (2009).

Conclusion:

Information about the physiology of pulpal pain and the sensory fibers causing this pain, together with information gathered from the patient, and the use of appropriate devices to test pulp sensitivity and vitality are very critical to reaching an accurate diagnosis on which to base an appropriate treatment plan. Multiple devices that test pulp viability are available on the market, but they test the viability of nerve fibers as measures of pulp vitality resulting sometimes in false positive or false-negative results. These can lead to unnecessary endodontic procedures if these tests are not substantiated with results of other diagnostic measures. Pulpal blood flow, which is at least as important as testing the neural supply of the pulp, must also be examined. Although still being studied, methods to test blood flow look very promising and should soon be in use in the dental clinic.

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